VOLUME LXIII AUGUST, 1953 NUMBER 8

THE LARYNGOSCOPE

FOUNDED IN 1896
BY
MAX A. GOLDSTEIN, M.D.

PUBLISHED BY
THE LARYNGOSCOPE
640 SOUTH KINGSHIGHWAY ST. LOUIS (10), MO., U.S.A.

NOTICE TO CONTRIBUTORS

THE LARYNGOSCOPE reserves the right of exclusive publication of all articles submitted. This does not preclude their publication in Transactions of various Societies.

Manuscripts should be typewritten, double spaced, on one side of paper only and with sufficient margins to allow for corrections.

References should be complete: author's surname, initials, title of article, Journal, volume, page, month, year.

Six illustrations will be furnished for each article without cost to author. Authors will please limit illustrations to six or assume the expense of additional illustrations.

Proofs will be submitted to authors for corrections. If these are not returned, articles will be published as corrected in this office.

Reprints will be furnished at the following prices:

WITHOUT COVER

	250 Copies	500 Copies	1000 Copies	2000 Copies
Four Pages	$11.75	$13.75	$18.25	$28.25
Eight Pages	23.25	29.25	40.50	61.50
Twelve Pages	34.75	45.50	66.00	104.00
Sixteen Pages	45.50	56.00	76.00	115.50
Twenty Pages	54.00	67.00	92.50	143.00
Twenty-four Pages	65.00	80.50	110.50	169.00
Twenty-eight Pages	76.75	93.50	127.00	193.00
Thirty-two Pages	86.00	104.00	140.00	212.00

WITH COVER

Four Pages	$22.25	$27.75	$38.00	$60.50
Eight Pages	33.75	43.25	60.25	93.75
Twelve Pages	45.25	59.50	85.75	136.25
Sixteen Pages	56.00	70.00	95.75	147.75
Twenty Pages	64.50	81.00	112.25	175.25
Twenty-four Pages	75.50	94.50	130.25	201.25
Twenty-eight Pages	87.25	107.50	146.75	225.25
Thirty-two Pages	96.50	118.00	159.75	244.25

Express charges will be paid by consignee.

THE
LARYNGOSCOPE.

VOL. LXIII AUGUST, 1953 No. 8

DEVELOPMENT OF OTOLARYNGOLOGY IN THE SOUTH.

PRESIDENTIAL ADDRESS.*

FRANCIS E. LEJEUNE, M.D.,
New Orleans, La.

It is with a deep feeling of humility that I express my genuine appreciation for the honor and privilege of serving as your president. The privilege of visiting the sectional meetings in the role of President is one of the most stimulating, interesting and enjoyable experiences that I have ever had. The courtesies extended and the good fellowship engendered are never to be forgotten.

It seems fitting that New Orleans should have been selected as the city where the fifty-seventh annual meeting of the American Laryngological, Rhinological and Otological Society would be held. The fifty-sixth annual meeting was held in Canada, another land of French settlers, one of whom journeyed down the Mississippi river in 1718 to found the settlement of "La Nouvelle Orleans." This French Canadian from Montreal was Jean Baptiste LeMoyne, Sieur de Bienville, who served as Louisiana's first governor. Moreover, this year New Orleans is celebrating the one hundred fiftieth anniversary of the Louisiana Purchase. It was in November 1803 that the territory of Louisiana was returned to France by Spain, which had acquired it from France in 1762 in a secret treaty. Twenty

*President's Address before the Fifty-seventh Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., New Orleans, April 28, 1953.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, June 22, 1953.

days after this exchange France sold this territory to the United States. Finally, one of the descendants of the Acadians, who in 1765 were exiled by the English from Canada to Maryland, Virginia and Louisiana, bids you welcome to the Deep South, especially to our city.

The story of the development of otorhinolaryngology into the specialty which we practice today could never be told without mentioning the names of numerous brilliant physicians listed in the roster of our Society. Time, however, will not permit me to give an account of even the most outstanding contributions of all of these members. For this reason, and also because we are meeting today in the Deep South, it seemed appropriate to pay tribute at this time to a few of those men who played an outstanding part in the development of otorhinolaryngology in the South.

One of them was Robert Clyde Lynch, of New Orleans. A born leader and profound thinker, he was a man of great charm whose untimely death deprived otolaryngology of a prodigious worker. The Lynch frontal sinus operation remains even today one of the best approaches to chronic infections involving the frontal sinuses. Dr. Lynch was probably best known for his contributions to laryngology; he perfected and popularized suspension laryngoscopy, which supplied considerable impetus to the development of intralaryngeal surgery. His lectures and clinics were largely attended, and he possessed the faculty of making difficult problems appear simple, the mark of a great teacher. A founder of the American Board of Otolaryngology, he exerted great influence on the development of otolaryngology in the South. Though destined for many honors in his chosen field, an accident claimed his life at the early age of 51. His influence on Southern otolaryngology is still keenly felt today.

Joseph Addison Stucky, of Lexington, Kentucky, will probably be best remembered for his efforts directed toward establishment of special clinics in the mountains of Kentucky for the treatment of trachoma and surgical diseases of the eye, ear, nose and throat. His passion for service and his great capacity for friendship are reflected in the many hours he and his

associates devoted to the treatment of these mountain people. Though he was a member of all national otorhinolaryngologic societies, the one dearest to his heart was our very own Society, which he served as President. His ability and skill attained national recognition, but he will long be remembered for his loyal friendship and kindly deeds.

Dunbar Roy, of Atlanta, Georgia, was known as one of the "Old Guard" in otolaryngology. A man of considerable means, he nevertheless was active in the practice not only of clinical but also of academic medicine. He was Professor of Otolaryngology at Emory University until ill health forced his resignation. He held high positions in all the national otorhinolaryngological societies and in 1923 he served as president of the American Laryngological, Rhinological and Otological Society. His numerous contributions to medical literature especially reflected his practical experience, sincerity and sound judgment. Affable, gracious and loyal, he was truly a gentleman of the old South.

Another member of the "Old Guard" was James Wilkinson Jervey, of Greenville, South Carolina. Member of a distinguished, scholarly southern family, he upheld the standards laid down by four consecutive generations of physicians. His leadership in medical organizations is reflected in the offices he held, culminated perhaps by his election to the presidency of our Society in 1933. Recognition of his ability as an executive outside of his own specialty came when he was elected president of the Southern Medical Association in 1937. He contributed many articles to the medical literature, particularly on clinical techniques in ophthalmologic and rhinologic surgery. A scintillating personality with a keen wit, he will long be remembered by those who knew him as one gifted in oratory and a splendid raconteur.

John Shea, of Memphis, Tennessee, was never so happy as when discussing family, religion and his chosen specialty. Gifted with a pleasing personality and a ready smile, he was an energetic and enthusiastic worker in otolaryngology, contributing many articles on various subjects. He was particularly interested in the welfare of the younger physicians and

was never too busy to discuss their problems. He encouraged many of them to enter the practice of otorhinolaryngology and with the simplicity and benevolence of greatness he was ever ready to extend to them a helping hand. His efforts in behalf of otolaryngology are perhaps best reflected in the offices he held in national otolaryngological societies. He served as president of our Society and was president-elect of the Academy of Ophthalmology and Otolaryngology. He was also a member of the American Board of Otolaryngology, where he worked unceasingly to keep our standards at the highest level. By his work, conduct and life he helped to raise the standards of otolaryngology in the South.

Because of these men southern otolaryngology has progressed and we are immeasurably richer for their having been with us.

In closing, I would like to point out to the energetic student possessed of initiative and vision the unexcelled opportunities which otolaryngology offers as a practicing specialty. It has broadened considerably with the years and now good otolaryngological training encompasses not only fundamental training in diseases of the ear, nose and throat, but also meticulous training in fenestration and other otitic surgery, bronchoesophagology, maxillofacial surgery, rhino-plasty, allergy and neck surgery.

With the advent of the chemotherapeutic and antibiotic agents we have been better able to control acute infections and their complications, and as a result of this successful therapy, other aspects of otolaryngology long neglected, are now receiving deserved attention. This means that new and interesting problems are being seen daily, which together with the forty-odd unsolved problems posed by Proetz a few years ago, are still a challenge to the skill, ingenuity and investigative minds of interested specialists. A specialty which offers as many unsolved problems and opportunities for continued study as does otolaryngology is not only an incentive but also beckons young physicians seeking an interesting and gratifying profession.

RE-EVALUATION OF SEMON'S HYPOTHESIS.*

LOUIS H. CLERF, M.D.,
and
WILLIAM H. BALTZELL, M.D.,
Philadelphia, Pa.

In 1881 Sir Felix Semon arrived at certain conclusions based on a study of 22 cases of paralysis of the larynx and an extensive review of the literature, and formulated a working hypothesis to explain the behavior of paralyzed vocal cords. This postulate has come to be known as "Semon's Law." It is our purpose to re-evaluate this work in the light of present day knowledge to determine its validity.

Semon[1] and Morrell Mackenzie had previously proclaimed that "the occurrence of an isolated paralysis of the abductor filaments of the recurrent nerve in cases in which the roots or trunks of the spinal accessory, pneumogastric and recurrent nerves are injured or diseased, is not an isolated pathological curiosity. There is a distinct proclivity of the abductor fibers to become affected, in such cases, either at an earlier period than the adductor fibers, or even exclusively." He presented clinical and postmortem data showing that isolated paralysis of the posterior cricoarytenoid muscles was the result of disease or injury to the centers and nerve trunks, or, that at any rate, the paralysis of these muscles was earlier and more developed than that of their antagonist.

He suggested several explanations, namely, "that activity of the abductors, although not entirely beyond control of the will, is much more automatic than that of the antagonist and perhaps its power of resistance against disease-producing causes is less" and that this "proclivity of the abductor cen-

*Read at the 57th Annual Meeting of the American Laryngological, Rhinological and Otological Society, New Orleans, April 30, 1953.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication May 4, 1953.

ters to succumb to central causes of disease is quite analogous to a similar proclivity of the extensor muscles of the extremities to become sooner affected than the flexors."

Several additional hypotheses were made to explain this peculiar proclivity, namely: the anatomical distribution of the fibers of the recurrent nerve might be a concentrical one and that the abductor fibers might be situated in the periphery of the nerve and most exposed to external injuries, that there might be a specific vulnerability of the abductor filaments and also, that possibly the abductors might receive an increment of nerve force from the superior laryngeal.

It is our contention that the hypothesis is not supported, in fact, by any one of these explanations and that the entire problem can be explained on an anatomical basis.

For many centuries, in fact from the time of Galen, it has been commonly accepted by authorities that the recurrent laryngeal nerve supplied all the intrinsic muscles of the larynx except the cricothyroid which was innervated by the external branch of the superior laryngeal nerve. Many clinicians believed, however, that the interarytenoid muscle received motor fibers from the superior laryngeal nerve. This was stated as early as 1884 by Exner although no anatomical evidence was presented to support it.

Much confusion has existed in the minds of laryngologists to explain anatomically the position of the vocal cords following injury or disease of the nerve trunk or centers. The work of King and Gregg,[2] which supported the anatomical studies of Toldt,[3] Piersol,[4] Weeks and Hinton,[5] Armstrong,[6] and others, clearly demonstrated that the midline position assumed by one or both vocal cords in certain cases of thyroidectomy could be explained on the basis of injury to the posterior subdivision of the recurrent nerve which had divided extralaryngeally. If the anterior subdivision, which supplies the adductor groups of muscles remained intact, being unopposed, it adducted the vocal cord bringing it to the midline.

Weeks, Hinton, and Armstrong found that the recurrent nerve divided extralaryngeally in 73 and 78 per cent of the

specimens examined. These studies carried out prior to King and Gregg's observations, as well as the more recent work of Morrison and Rustad,[7] have amply corroborated Toldt's original findings that the recurrent nerve often divides extralaryngeally.

The observations made by laryngologists in many cases of abductor paralysis following thyroidectomy and involving one or both vocal cords over a period of years have shown conclusively that the appearance and the position of the cords remained unchanged. We have observed eight cases of bilateral abductor paralysis, occurring after thyroidectomy that had existed for from 20 to 33 years and many for periods less than 20 years. There certainly was no tendency toward any appreciable change in the position of the cords during this time. It is our opinion that this form of paralysis does not come within the purview of Semon's hypothesis and can be explained on the basis of injury only to the fibers of the recurrent laryngeal nerve that supply the abductor muscle.

This, however, does not explain the paramedian position which is promptly assumed by a vocal cord following injury or disease of the entire recurrent nerve.

Interest in this entire subject was stimulated by Todd, who, in 1938, demonstrated motor end plates on the internal branches of the superior laryngeal nerve in the interarytenoid muscle of man. The work of Lemere,[8] which had been done exclusively on dogs but which was accepted as applicable to man showed that there were no motor end plates from the superior laryngeal nerve to the interarytenoid muscle.

In 1952, Vogel,[9] used an improved technique and corroborated Todd's work. He demonstrated conclusively that there were motor end plates in the interarytenoid muscle connected to the internal subdivision of the superior laryngeal nerve in man. He repeated the work previously done by Lemere and corroborated his findings, namely that there was no motor fibers distributed to the interarytenoid muscle of the dog. In his studies, Vogel found that in all cases in the human larynx, but never in dogs, fibers of the internal laryngeal branch were traced to the motor end plates in the interarytenoid muscle.

The results of dissection and histologic studies showed that in man the internal laryngeal nerves are not purely sensory as they are in the dog but have a motor component that innervates the interarytenoid muscle. He also demonstrated that this muscle, too, is innervated by the recurrent laryngeal nerve.

It would appear, therefore, that the double innervation of the interarytenoid muscle would explain the paramedian position of the vocal cord following injury to the entire recurrent nerve. This had previously been ascribed only to the action of the cricothyroid muscle, a tensor and adductor innervated by the external branch of the superior laryngeal nerve.

The explanations advanced by Semon might at this time be considered. No definite evidence is available to indicate that there is lessened resistance of the abductor muscles to diseases. Negus[10] suggested that phylogenetically the adductors or constrictors were earlier and thus might be more resistant to disease. While there may be some truth in this, it is far overshadowed by the fact that there is double innervation of the interarytenoid muscle.

The second premise that the proclivity of the extensor muscles of the extremities are more prone to disease than the flexors might be applied to the laryngeal muscles probably is not valid. Primarily, the muscles of the larynx are really constrictors and dilators and not flexors and extensors in the true sense of the term. Fulton[11] stated that the "flexor reflex is the most primitive pattern of response of higher vertebrates, representing the mechanism for withdrawing an extremity from injury while the extensor reflex is primarily concerned with resisting the action of gravity." The action of the larynx seems to have little to do with these.

The idea of a possible concentrical arrangement of the recurrent fibers and the occurrence of the abductor fibers near the periphery and, therefore, more subject to injury, has been refuted by Murtagh and Campbell[12] who found no exclusively peripheral distribution of abductor fibers.

Clinical experience has shown that complete section of a recurrent occurring during pneumonectomy or esophagectomy

results in immediate complete paralysis of the corresponding vocal cord[13]. The arytenoid assumes a paramedian position being slightly abducted and tilted forward and there is bowing of the membranous portion of the cord with slight prominence of the tip of the vocal process. A number of these cases have been observed for from several months to more than two years after their occurrence and during this interval no appreciable change in the appearance of the paralyzed cord was noted except that there was increased compensatory adduction of the normal cord.

There also have been observed seven cases of paralysis of the left vocal cord following a single-stage combined diverticulectomy. This was attributed to compression of the corresponding recurrent nerve by a metal retractor. These patients all were hoarse, and the larynx, which was examined within 24 to 48 hours after operation, revealed the left vocal cord in a paramedian position with loss of tension. Complete recovery of motility and a normal voice resulted within four to six months in all cases.

It would appear that if the abductor fibers of the recurrent nerve exhibited a proclivity to be affected earlier or more readily to injury, either because of their location in the common nerve trunk or because of their smaller size, the paralyzed vocal cord should first have been in a position of adduction.

In the cases of paralysis ascribed to mechanical causes, namely, aneurysm, cardiac enlargement, pulmonary tuberculosis and anthrasilicosis, one rarely sees the involved cord in a position of adduction. The causes in this category are progressive and result from stretching or compression.

Similar comment may be made in extralaryngeal neoplastic cases associated with laryngeal paralysis. The cause here is direct invasion or compression of the nerve and is progressive. In 96 cases of carcinoma of a bronchus, trachea, or esophagus and mediastinal metastasis from distant lesions, the paralyzed vocal cord was in a paramedian position with loss of tension, and hoarseness was a prominent symptom.[13]

Many of us have erroneously indicated that the paralyzed cord in these groups of cases was in a cadaveric position. The "cadaveric position" is assumed by a vocal cord after complete interruption of the recurrent and superior laryngeal nerves. Negus[14] suggested that this be called "combined paralysis" to indicate that both laryngeal branches of the vagus are involved, and we are in accord with this view. The position of the cord in the cadaveric position is an intermediate one between abduction and adduction with forward tilting of the arytenoid, loss of tension of the cord and prominence of the tip of the vocal process of the arytenoid. These patients exhibit no dyspnea, have impairment of the voice and are unable to cough in a normal manner. There is air wastage.

Reference has been made to the action of the cricothyroid muscle, a tensor of the vocal cord and adductor of the arytenoid, and the assumption that it was responsible for maintaining the paramedian position of the cord in recurrent nerve paralysis. As has been shown in paralysis of the posterior and lateral cricoarytenoid muscles which occurs in complete interruption of a recurrent nerve, the position of the arytenoid no longer is maintained on the cricoid cartilage by the splinting action of these muscles and the joint capsule. The arytenoid is tilted forward, and the distance between it and the anterior commissure is shortened. The cricothyroid muscle, therefore, is no longer able to influence the position of the arytenoid.

In conclusion, therefore, we believe that acceptance of the findings of Todd and Vogel, that there is double innervation of the interarytenoid muscle from the inferior or recurrent nerve as well as the internal subdivision of the superior laryngeal nerve, and of the application by King and Gregg of Toldt's observation that the recurrent nerve may divide extralaryngeally, will explain the position of the vocal cord following injury or disease of a recurrent nerve or its center. Semon's postulate and the other hypotheses appear inadequate and unnecessary.

REFERENCES.

1. SEMON, F.: On the Proclivity of the Abductor Fibres of the Recurrent Laryngeal Nerve to Become Affected Sooner than the Adductor Fibres, *Arch. Laryng.* 2:197 (July 1) 1881.

2. KING, B. T. and GREGG, R. L.: An Anatomical Reason for the Various Behaviors of Paralyzed Vocal Cords. *Ann. Otol., Rhinol. and Laryng.* 57:925 (Dec.) 1948.

3. TOLDT, C.: An Atlas of Human Anatomy, p. 881. Rebman Co., N. Y. 1904.

4. PIERSOL, G. A.: Human Anatomy. J. B. Lippincott Co., 1907.

5. WEEKS, C. and HINTON, J. W.: Extralaryngeal Division of the Recurrent Laryngeal Nerve. Its Significance in Vocal Cord Paralysis. Ann. Surg. 116:251 (Aug.) 1942.

6. ARMSTRONG, W. G. and HINTON, J. W.: Multiple Divisions of the Recurrent Laryngeal Nerve. An Anatomic Study. Arch. Surg. 62:532 (April) 1951.

7. RUSTAD, W. H. and MORRISON, L. F.: Revised Anatomy of the Recurrent Laryngeal Nerves. THE LARYNGOSCOPE. 62:237 (Mar.) 1952.

8. LEMERE, F.: Innervation of the Larynx. Innervation of Laryngeal Muscles. *Am. J. Anatomy.* 51:417 (Nov.) 1932.

9. VOGEL, P. H.: The Innervation of the Larynx of Man and the Dog. *Am. J. Anatomy.* 90:427 (May) 1952.

10. NEGUS, V. E.: The Mechanism of the Larynx. Wm. Heineman, Ltd., London, 1929.

11. FULTON, J. F.: Physiology of the Nervous System, 3rd ed. Oxford Univ. Press. 1949.

12. MURTAGH, J. A. and CAMPBELL, C. J.: Physiology of Recurrent Laryngeal Nerve. *J. Clin. Endocrinol. and Metabolism.* 12:1398 (Oct.-1952.

13. CLERF, L. H.: Unilateral Vocal Cord Paralysis. *J.A.M.A.* 151:900 (Mar. 14) 1953.

14. NEGUS, V. E.: Certain Anatomical and Physiological Considerations in Paralysis of the Larynx. *Proc. Royal Soc. Med* 40:849 (Dec.) 1947.

BRONCHOGENIC CARCINOMA. THE ROLE OF THE BRONCHOSCOPIST IN ITS EARLY DIAGNOSIS.*

ALTON OCHSNER, M. D. (by invitation)
New Orleans, La.

I am very happy to talk to you about bronchogenic carcinoma and speak about the role of the bronchoscopist in this disease. This is a disease which should interest all of us because it is increasing more than any other cancer in the body. One of the reasons I think otolaryngologists should be interested in this subject is that it appears that otolaryngologists frequently maintain cigarettes cause no irritation of the respiratory tract. Although there has been some controversy concerning the tremendously increased incidence of cancer of the lung, I am convinced that it is due to the increased use of cigarettes. There is a distinct parallelism between the use of cigarettes and the incidence of bronchogenic cancer. In 1920 bronchogenic cancer represented 1.1 per cent of all cancers; in 1930, 2.2 per cent of all cancers; in 1949, 8.3 per cent of all cancers, and we predict (although I am not Drew Pearson) that in 1970, 18 per cent (approximately one in every five) of all cancers will be bronchogenic.

From 1920 to 1948 the death rate in the United States from bronchogenic cancer increased over ten times (1.1 per hundred thousand population in 1920 to 11.3 per hundred thousand in 1948). Whereas cancers generally have increased, 31 per cent in the last 20 years, the increase in the incidence of bronchogenic cancer has been 144 per cent.

The reason why cancers generally are increasing is that we are living to an older age and cancer is a disease primarily of

*Read by invitation at the Fifty-seventh Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., New Orleans, La., April 28, 1953.

†From the Department of Surgery, School of Medicine, Tulane University of Louisiana and the Ochsner Clinic, New Orleans, La.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, July 7, 1953.

older people, but this will not explain the exceptional increase in the incidence of bronchogenic carcinoma. There is a definite parallelism between age and the development of cancer. All cancers of the body except bronchogenic cancer increase with advancing age, both in men and women. In each decade beyond forty years of age the incidence of cancer increases; however, the incidence of bronchogenic carcinoma does not increase progressively up to the age of ninety but increases more rapidly than other cancers up to the age 65 in men and 70 in women. After these respective ages, the incidence decreases somewhat in men and remains approximately the same in women. This difference in the incidence in lung cancer might be due to one of two factors. It is probable that people 70 years of age and older do not and never have smoked as much as younger people do today and, therefore, the older persons have been subject to less carcinogenic effect of tobacco. On the other hand the decrease in the incidence of bronchogenic cancer in older persons may be due to the fact that the heavy smokers have developed vascular diseases particularly coronary thrombosis and have succumbed before the carcinogenic effect of the smoking has had a chance to produce its damage.

There is a distinct parallelism between the sale of cigarettes in the United States and the incidence of bronchogenic cancer. Dr. Evarts Graham, for the past two years, has been conducting an investigation concerning the carcinogenic effect of tobacco tar. Employing a robot which smokes cigarettes in the same manner in which a human being does he has secured a tar-like residue which when applied to the epithelial surfaces of animals produces cancer similar to human cancer in 40 per cent of the animals. Both Dr. Graham and I believe the percentage of "positive takes" will increase as the length of time increases because it is well known that a definite time period is necessary for a carcinogen to cause cancer.

In New Orleans we have an ideal opportunity to determine the racial incidence of cancer or any other disease because in the Charity Hospital white and negro patients are ad-

mitted with about equal frequency. Bronchogenic cancer is primarily a disease of the white people, 172 per 100,000 as compared to 124 in the negroes.

Bronchogenic carcinoma is also a disease primarily of men. In our series of cases 90 per cent were men. Dr. Evarts Graham is of the opinion that the incidence will increase in women as much as it has in men. I don't believe that it will because I believe that there is a sexual predisposition to bronchogenic cancer in men and that it will always be more frequent in males.

The type of neoplastic lesion varies with the two sexes. In our series in the men, 57 per cent of the lesions were epidermoid, 28 per cent were undifferentiated and 15 per cent were adenocarcinomas. In the women, 22 per cent were epidermoid, 28 per cent undifferentiated and 50 per cent adenocarcinoma. We believe that adenocarcinoma in the bronchus develops on the basis of an epithelial rest, which is a congenital remnant and that most epidermoid carcinoma of the bronchus are probably caused by the carcinogenic agent in cigarette smoke.

Of all our cases, almost 53 per cent were epidermoid. Twenty-eight per cent were undifferentiated and only 19 per cent adencarcinoma. Of all the epidermoid lesions, 95 per cent were in men and only five per cent in women; of the undifferentiated, 88 per cent were in men and 12 per cent in women; of the adenocarcinomas 68 per cent were in men and 32 per cent in women.

As is well known, carcinoma of the bronchus is a disease primarily of advancing age. The greatest incidence (38 per cent) is in the sixth decade, followed in frequency by the seventh (30 per cent) and fifth (19 per cent) decades; however, the two extremes of life are not immune.

A fact of importance to laryngologists and bronchoscopists is that bronchogenic carcinoma is a disease primarily of the upper lobes. In 56 per cent of our cases the upper lobes were involved, 27 per cent in the right upper lobe, 29 per cent in the left upper lobe, and it is for this reason that

these lesions may not be visible on bronchoscopy. The two lungs are involved with about equal frequency, the right one (54.4 per cent) being somewhat more frequently involved than the left one (45.6 per cent).

Also of interest to bronchoscopists is the relative location of the tumor in the bronchus. There was a time when I thought that most bronchogenic carcinomas were located in the main stem bronchi; however, this is not so. In our cases, the main stem bronchus was involved in only four per cent, the lobe bronchus in 60 per cent and the peripheral bronchus, 36 per cent. Of the epidermoid carcinomas, only 68 per cent were in lobe bronchi, four per cent in the main stem bronchi, and 28 per cent in the periphery. Of undifferentiated lesions, three per cent were in the main stem bronchi, 53 per cent in lobe bronchi and 44 per cent in the periphery. Of the adenocarcinomas, five per cent were in the main bronchi, 46 per cent in the lobe bronchi and 49 per cent were in the periphery. It has been known for some time that adenocarcinomas occur more frequently in the periphery than elsewhere.

As mentioned previously we are of the opinion that adenocarcinomas develop from a congenital rest, which contention is substantiated by decrease in the incidence of adenocarcinoma with advancing age in contradistinction to epidermoid carcinoma which increases with advancing age. Up to the age of 50, adenocarcinomas and epidermoid carcinoma occur with about equal frequency. After that age the adenocarcinomas tend to decrease in incidence. In other words, an individual who has a congenital rest, from which adenocarcinoma develops, is likely to develop it early in life. On the other hand, in an individual whose malignant lesion is the result of a carcinogenic agent, the longer that an agent is used the greater the chance of malignant change. We feel that the incidence of epidermoid carcinoma with advancing age is due to the longer effect of the carcinogenic agent in cigarette smoke.

Unfortunately, there are no classical clinical manifestations of bronchogenic cancer. In over half of our cases, there

was a history of a previous respiratory tract infection. This point cannot be emphasized too much although its emphasis to otolaryngologists is probably less important than to general practitioners who first see these patients. In the past year it has been particularly distressing to us that there has been a longer delay from the onset of the first symptom until definitive therapy is instituted than there was five years ago. I think this is due to the fact that about five years ago there was a good deal in medical literature about viral pneumonia which was correctly described as an atypical pneumonia. Unfortunately the medical profession has come to think of atypical pneumonia and viral pneumonia as being synonymous. Probably because of this too frequently a patient with atypical pneumonia is diagnosed as having viral pneumonia. The patient is treated for weeks and months with some improvement, to be sure, because the administration of antibiotics will control to a certain extent the pneumonitis associated with a bronchogenic cancer. Whereas, I am sure that a man past 40 can develop a viral pneumonia, I firmly believe that such an individual who has been a heavy smoker is more likely to have bronchogenic carcinoma. It must be emphasized that in all such individuals whose pneumonitis does not respond quickly and completely subside within a few weeks, the possibility of bronchogenic carcinoma must be considered and diagnostic procedure instituted to confirm or exclude such a diagnosis.

Cough is the principal manifestation of bronchogenic cancer, but because most smokers have a cough it is likely to be disregarded. Hemoptysis always needs investigation because it is the result of ulceration. Thoracic discomfort is of importance; because most of us are not conscious of the fact that we have a thorax and if at any time we do become conscious of it, it is indicative of some abnormality. Also wheezing, particularly in an individual who has not wheezed previously, demands investigation because wheezing is an indication of bronchial obstruction. If the patient has had asthma all his life wheezing is of less importance because it is probably caused by bronchial spasm and not by neoplasm. On the other hand, in a man past 40, who has not had

asthma and wheezed previously, but who for the first time begins to wheeze, the bronchial obstruction is usually the result of cancer.

The late manifestations of bronchogenic carcinoma are those of complications. Fever, may be due to associated pneumonitis caused by obstruction of a bronchus by the tumor or to necrosis of the tumor with the production of an abscess. Pain is usually due to involvement of the parietes by the tumor. Digestive disturbances usually result from vagus nerve involvement. Emaciation is the result of extensive disease and infection.

In our resected series cough was present in 89 per cent, weight loss in 70 per cent, chest pain in 66 per cent, hemoptysis in 55 per cent, a previous respiratory tract infection in 54 per cent, dyspnea in 51 per cent, and voice change in seven per cent. I think it is important to discuss the significance of voice change in patients with bronchogenic carcinoma which is due to involvement of a recurrent laryngeal nerve, because there are many, including thoracic surgeons, who believe that recurrent or phrenic nerve involvement is indicative of inoperability.

Whereas involvement of either or both of these nerves means extension of the neoplastic process beyond the lung, we are convinced that it does not always mean non-resectability, although it must be admitted that a large number of such patients might not be salvaged. We have not accepted recurrent and/or phrenic nerve involvement as evidence of inoperability and have performed exploration in such cases unless there was other evidence of inoperability. In seven per cent of our patients in whom a resection was done there was involvement of the recurrent laryngeal nerve. In many, the operation was only palliative but the patient has a more comfortable existence because the suppurating lung which was producing the patient's symptoms was removed. We have one patient who has survived six and one-half years and had that patient not been operated, even though his recurrent laryngeal nerve was paralyzed, I am sure he would be dead today. We feel that although the prognosis is worse

in these individuals we are not justified in denying them the chance of exploration simply because they have recurrent laryngeal or phrenic nerve paralysis.

The diagnosis of bronchogenic cancer isn't difficult generally if one will only think of the possibility of its existence. Roentgenography is one of the best methods of making the diagnosis because of the ease with which the X-ray can be obtained. When the tumor is large enough, a shadow can be demonstrated on the X-ray film. A small tumor which only incompletely obstructs the bronchus can produce obstructive emphysema. Because of the dilatation of the bronchus during inspiration, air can get beyond the tumor which does not completely obstruct it, whereas during expiration due to contraction of the bronchus it becomes occluded by the tumor and obstructive emphysema distal to the tumor results. Atelectasis is telltale evidence of bronchial obstruction, because, following complete obstruction of a bronchus, air distal to the obstruction is absorbed from the bronchial tree. Bronchography is of value in the cases in which the lesion is in the upper lobe or in the periphery areas which may not be visible bronchoscopically.

Bronchoscopy of course, should be done in every instance, although in our cases, we were able to make a positive diagnosis on bronchoscopy in only 37 per cent of instances, which probably was due to the fact that over half of the tumors were located in the upper lobes and about a third in the periphery. Bronchoscopy is of importance not only because the lesion frequently can be visualized and biopsied but also to obtain bronchial secretions and to determine the condition of the trachea and main stem bronchi. The last finding is important to determine inoperability in some cases.

The method of diagnosis which gave us the highest percentage of positive diagnosis was cytological examination of the bronchial secretions. In 68 per cent of our cases, we were able to make a positive diagnosis by cytologic examination of bronchial secretion or the sputum. I don't think it makes much difference whether these are obtained during bronchoscopy or by expectoration. Specimen for specimen

a higher incidence of positive diagnosis will be obtained on bronchoscopically aspirated secretions. On the other hand, because one can examine many more specimens of sputum than one is justified in obtaining by bronchial aspirations, more positive diagnoses are made in sputum examination.

Thorascopy is not of as much diagnostic as prognostic importance. We believe that in the case in which there might be extension from the lung to parietes, thorascopy is of value. Aspiration biopsy is mentioned only to condemn it. We feel it should not be used in a patient in whom there might be some chance of a cure. It is a valuable method of making a diagnosis of cancer in a patient with a peripheral lesion which is inoperable but should be avoided otherwise. We have seen four patients in whom implants along the course of the aspirating needle occurred.

A final method of diagnosis is thoracic exploration because using all the above prescribed diagnostic procedures of preoperative positive diagnosis of bronchogenic carcinoma can be made in only 80 per cent of cases.

The therapy of bronchogenic carcinoma is surgical extirpation. At the present time there are a number of thoracic surgeons who advocate lobectomy in the treatment of bronchogenic cancer. Whereas, I am sure that some bronchogenic cancers can be cured by lobectomy, I am convinced that it is an incomplete cancer operation. I feel about bronchogenic cancer just as I feel about any other cancer, that one cannot compromise with it. Whereas the statistics which are reported by Churchill and others are quite good following lobectomy, I am sure their results would be very much better had they used pneumonectomy in these cases. We do lobectomies, but we feel that lobectomy should be reserved only for the patient in whom the previous pulmonary functional studies indicate that a pneumonectomy can't be tolerated. One can't do a satisfactory cancer operation by doing a lobectomy because a complete operation, as I need not tell you, is one that consists of en bloc removal of the primary focus and all the regional lymph nodes which can be accom-

plished only by doing a pneumonectomy and complete en bloc excision of mediastinal lymph nodes. Because of this the best curative procedure for bronchogenic cancer is pneumonectomy.

In a series of 1122 cases of proven bronchogenic cancer which we have seen, 43 per cent were obviously inoperable when first seen. Six hundred and thirty-nine were considered operable, 57 (nine per cent) of whom refused surgery. Five hundred and eighty-two (91 per cent of the group considered operable and 52 per cent of the entire group) were explored, and 36 per cent were found to be nonresectable. We believe that if the lung containing the disease can be removed it should be. Of this group, 18 per cent died within the hospital. In 372 patients a pneumonectomy was performed (33 per cent of the entire group and 64 per cent of those explored) with a 20 per cent hospital mortality; 296 patients left the hospital alive (26 per cent of the entire group). Of our entire series, 33 per cent had resections and 48 per cent were so far advanced that exploration was considered hopeless. In the private series these respective percentages were 43 per cent and 34 per cent, as contrasted with 20 per cent and 66 per cent, respectively, in the Charity Hospital series.

Unfortunately there is too frequently a tragic delay between the onset of symtoms and the institution of definite curative therapy. There was an average delay of 8.7 months in the entire series, which has decreased somewhat recently with the exception of the past year during which time the delay has actually been longer than it was five years previously.

There are two possible sources of delay both of which are probably largely avoidable: one is that for which the patient is responsible; and the other for which the attending physician is responsible.

The total delay in the private cases was 9.3 months as contrasted with 7.5 months in the Charity cases. In the entire group the patient was responsible for a delay of 2.8

months and the physician for 5.5 months. These respective periods of time in the private cases were 2.6 months and 6.2 months and in the Charity cases were 3.0 months and 3.6 months. The longer delay caused by physicians in private cases is probably due to the fact that private patients were able to afford hospitalization and prolonged therapy for viral pneumonitis whereas charity patients unable to afford such prolonged hospitalization were referred to the Charity Hospital early in the course of the disease.

Because we physicians are responsible for a long delay in instituting definite therapy in bronchogenic cancer, it behooves us always to consider bronchogenic carcinoma in all heavy smoking men 40 years of age and older with thoracic symptoms. That delay is not the only factor responsible for inoperability is illustrated by the fact that the delay in the resected cases was 10.5 months and in the nonresected cases 7.5 months. Undoubtedly, the rapidly growing tumor produces symptoms relatively early and the diagnosis is suspected and confirmed fairly early. On the other hand the slow growing neoplasm producing minimal manifestation for many months is probably still resectable because of the slow growing neoplasm. Similar results have been reported by Rigler, O'Loughlin, and Tucker* who found that the average duration of symptoms in a nonresectable group of lung cancers was 13.1 months as contrasted with that of 19.4 months in a group of resectable cases.

In 74 per cent of our series the lesion had extended beyond the lung and the resection was classified as a palliative procedure, although in some, cures were obtained. In only 26 per cent was the lesion limited to the lung.

Of the various supplementary surgical procedures necessary to perform a pneumonectomy in about ten per cent it was necessary to ligate the vessels within the pericardium; four per cent had resection of a major portion of the chest wall, 3.5 per cent had a suture of the atrium because the tumor extended so far medially, a major portion of the diaphragm

*Rigler, L. G.; O'Loughlin, B. J.; and Tucker, R. C.: The Duration of Carcinoma of the Lung, Diseases of the Chest, 23:50, 1953.

was resected in 3.2 per cent and in one case a partial excision of the vena cava was done in order to remove a tumor. I mention these merely to demonstrate that whenever it is possible for us to get the tumor out we do so, because generally the patient's symptoms are due to the infection. Although this might be a palliative resection and they may die subsequently of their disease, at least the remaining days of their lives are comfortable. The overall mortality rate following pneumonectomy for bronchogenic carcinoma was 20 per cent. Prior to 1941 it was 48 per cent, from 1942 to 1946 it dropped to 20 per cent and in the 1947-52 period it was 18 per cent.

The mortality rate also depends upon the age of the patient. In our series it was 14 per cent up to 50 years of age; 50 per cent from 50 to 60 years, and 24 per cent above 60.

Of the various causes of death, 45 per cent were due to cardiovascular disorders. These probably can never be eliminated because most of the patients with bronchogenic carcinoma are in the sixth and seventh decades of life at which time cardiovascular accidents are prevalent. Thirty-two per cent were due to respiratory complications, 16 per cent to hemorrhage, and eight per cent were due to miscellaneous conditions.

In the consideration of the results obtained by treatment in patients with malignant disease one is interested not only in the operative survival rate but also the long term survival. In normal individuals in the same groups as bronchogenic cancer, the five year survival rate is 85 per cent.

In our series of bronchogenic cancer, the five year survival rate for the entire series whether they were operated upon or not was six per cent. In the entire group in which a resection was done the five year survival rate was 15.2 per cent. In those in which only a palliative resection was possible the five year salvage was 7.2 per cent, and in the cases in which the lesion was apparently limited to the lung, it was 36.2 per cent. In the cases in which no resection was done all of which were treated by other palliative measures

such as nitrogen mustard, X-ray therapy or a combination of both no patient survived as long as four years. This experience demonstrates that the only curative treatment of bronchogenic carcinoma is radical extirpation of the involved lung and the regional lymph nodes.

In conclusion, I would like to emphasize again that this is a disease which is increasing more rapidly than any other cancer. You, as otolaryngologists, have a real responsibility because of the large number of smokers with a cough and throat irritation who consult you. I know many of you smoke. I don't imagine any of you will discontinue the habit because of what I have said today, but I would urge that each of you who smokes get an X-ray of the chest every six months, or preferably every three months so that when you do develop your cancer it can be detected at the time when it is still localized. It is extremely desirable to make a diagnosis of bronchogenic carcinoma early, even before symptoms develop. In our series in which only six were symptomless, resection was possible in only 33 per cent, and the lesion was limited to the lung in only 8.5 per cent as contrasted with the finding in a small series of cases seen by Overholt in which the malignant lung lesion was diagnosed before symptoms developed. In all these cases the lung was resectable and in 75 per cent the lesion was limited to the lung.

AN APPARENT PRIMARY CHOLESTEATOMA* CASE REPORT.

HOWARD P. HOUSE, M.D.,
Los Angeles, Calif.

A 26-year-old female stated she had had constant vertigo over a period of some three years. Complete examinations had been made by competent neurologists without finding definite evidence of intracranial pathology. The patient had noted a feeling of fullness with associated tinnitus in her left ear during the past six months and more recently became incapacitated due to vertigo.

Examination revealed a slight nystagmus with the slow component directed toward the left. Examination of the ear failed to reveal any local evidence of pathology and the ear drum appeared essentially normal. The nose and throat were essentially negative.

Caloric studies revealed a marked hyperactive response in the left ear and a normal response in the right ear. Audiometric tests revealed normal hearing in the right ear and a 30 db loss in the left ear by air conduction.

In view of this patient's incapacitating vertigo and since all other examinations had been essentially negative, it was decided to explore the left ear and possibly destroy the labyrinth, in spite of good cochlear function, if no other cause for the vertigo could be found.

An endaural approach was made in the usual manner and the mastoid and epitympanum appeared to be essentially normal. When the incus was removed it was found the long process had been destroyed and an oval, white pearly substance about 4 mm. in diameter was occupying the stapes region. A

*Presented as a motion picture demonstration at the meeting of the Western Section of the American Laryngological, Rhinological and Otological Society, Los Angeles, January 24, 1953.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, May 17, 1953.

radical mastoidectomy was performed and a completely encapsulated, small, pearl-like cholesteatoma was removed from the oval window area. The long crus of the incus, the stapes and the stapes footplate had been destroyed.

Since surgery, the patient has been free of the vertigo, which was apparently produced by the cholesteatoma causing pressure in the oval window area.

Comment: This case presents the unusual phenonomen of an apparent primary cholesteatoma occupying the area of the stapes.

LARYNGEAL STENOSIS.*†‡

DEGRAAF WOODMAN, M.D.,
New York, N. Y.

In the course of surgery for bilateral abductor paralysis, we have been asked to see a number of cases which had had previous attempts at corrective surgery on both sides of the larynx, in which decannulation of the patients was not accomplished. I am sure, therefore, after having discussed this problem with others doing this work, that there are a few score of these individuals who need further help.

Until this year there were six such patients who had permitted me to carry on still further surgery to relieve them of wearing their tracheotomy tubes. Only three or fifty per cent of these resulted in decannulations. We felt that considering the scar tissue and contractures from previous operative work, perhaps this was a pretty good average.

This year a case came to us with a particularly complicated background of previous surgical failures and the procedure we carried out terminated in such a good result, that I think it offers a chance of a much higher percentage of successes in the rehabilitation of these problem cases; hence this report.

The patient, a 38 year old housewife, gave a history of dyspnea on the slightest exertion, and hoarseness which immediately followed a thyroid operation for goiter in 1947. The hoarseness disappeared after two weeks, but the dyspnea persisted. One year later a tracheotomy became necessary. In 1949 an attempt was made to do a right arytenoidectomy. The operators started to do an arytenoidectomy via a window in the thyroid ala, but due to edema and inability to visualize the larynx with the laryngoscope, they ended up by placing a suture around the arytenoid, tying it to the thyroid cartilage. This failed to permit removal of the tube. In 1951 a left cordectomy via laryngofissure was unsuccessful. This was

*Read at the 57th Annual Meeting of the American Laryngological, Rhinological and Otological Society, New Orleans, La., April 28, 1953.

†From the Department of Otolaryngology, Presbyterian Hospital and College of Physicians and Surgeons, Columbia University.

‡Research supported in part by the Hayden Coakley Memorial Fund.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, May 4, 1953.

followed by the development of granulations at the site of the cordectomy, and an attempt to remove these via the laryngoscope resulted in producing a laryngeal web.

When first seen by me in April, 1952, she was wearing a No. 7 tracheotomy tube, which she was unable to cork even partially without developing marked dyspnea. Her voice was a whisper in character and was interrupted by breathy respirations and the need of placing her finger over the tube during expiratory phonation. She presented scars of previous surgery in the midline and right side of neck. Locally the larynx showed (Fig. 1) both cords to be in the paramedian position. The true cords had little or no motion on adduction or abduction. The false cords were hypertrophic and had some adductor powers but no abductor abilities. The right arytenoid area appeared to be tipped slightly forward. The glottic chink was closed in its anterior half by a web. A three-eighths inch anterior posterior slit was visible in the posterior aspect of the glottic chink. The patient was advised that we thought we might be able to decannulate, and we outlined our plan. She returned to her home town to think it over.

Fig. 1. Pre-operative Indirect View of Larynx.

On July 28, 1952, she was admitted to the Columbia-Presbyterian Medical Center for the purpose of attempting decannulation. At this time the larynx was inspected and was the same as when seen in April, except that the web had progressed even further posteriorly and there was now less than one-fourth inch anterior posterior slit-like aperture visible. Her general physical examination presented normal findings aside from the local laryngeal condition and she was pronounced satisfactory for operation. Her serum calcium, phosphorus, and cholesterol were all within normal range. Voice recordings were made.

Procedure

In this case the additional complication of a web gave us the opportunity to take advantage of McNaught's[1] Tantalum keel technique. Even with the web problem disposed of, the patient would still have cords which remained in the midline. This did not offer sufficient airway for decannulation. This meant that some further plastic procedure had to be carried out to gain more space. Her left cord had been at least partially

removed but was now a heavy band of scar tissue filling the left half of the glottic space. The right cord on which an arytenoidectomy had been attempted was still intact. This cord, therefore, offered us our best means of gaining additional airway.

The following steps were carried out:

1. Under general anesthesia, the midline of the larynx was exposed. A small vertical incision (Fig. 2-A) was made in the crico-thyroid membrane. A 1" circular saw was then used to cut the laryngeal cartilage in the midline from the crico-thyroid membrane incision up to about ¼" below the thyroid notch. Keeping the thyroid cartilage intact in this region prevents sliding and malalignment from developing when closure is attempted.

2. The web (Fig. 2-B and C) was then carefully cut with a No. 15 BP blade, keeping in the midline under the guidance of an assistant observing through a laryngoscope.

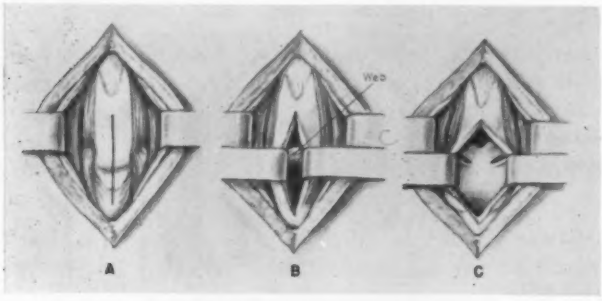

Fig. 2. (A) Midline incision starting in crico-thyroid membrane. (B) Exposure of web. (C) Web cut.

3. A mucosal flap was developed over the right vocal cord (Fig. 3-D) starting about one-eighth inch posterior to the anterior commissure and cutting posteriorly just above and a little below the cord, elevating the flap with its hinge (Fig. 3-E) attachment at about the level of the vocal process.

4. The mid two-thirds of the cord was then resected (Fig. 3-F), taking care not to cut into adjacent muscle layers deep to cord.

5. The mucosal flap was then reapplied (Fig. 4-G) to the depressed space on the lateral wall of the larynx and held in place with a 00 Deknatal suture. This was carried (Fig. 4-G and H) through the lateral thyroid ala cartilage with a Reverdin (2) needle, entering the larynx above and posterior to the mucosal flap and bringing the suture out again through the lateral wall of the larynx anteriorly and just below the flap, tying it outside (Fig. 4-I) the thyroid ala cartilage. This snugly applies the flap to the lateral wall thus insuring against the development of granulations and at the same time helps in gaining maximum glottic space by lateralization of the submucosal soft tissues (vocalis and thyroarytenoideus muscles).

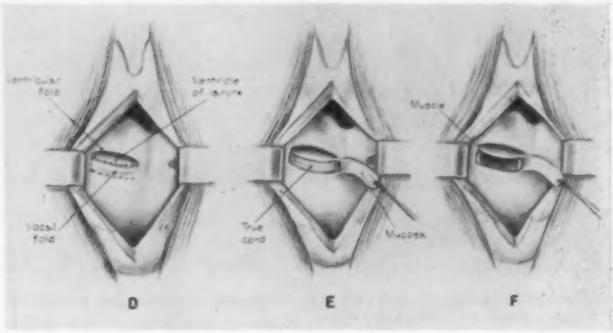

Fig. 3. (D) Flap of mucosa cut over right vocal cord. (E) Flap Hinge in region of vocal process. (F) Mid two-thirds of cord resected.

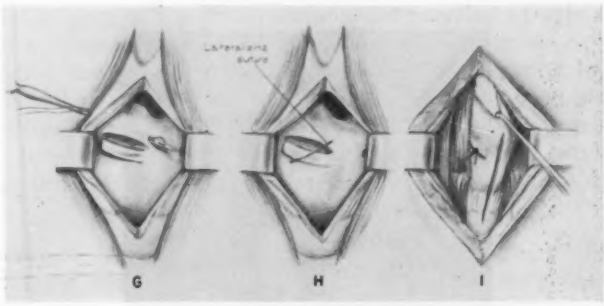

Fig. 4. (G) Flap reapplied to lateral wall. (H) Flap lateralized with suture placed with Reverdin needle. (I) Suture tied outside of ala cartilage.

6. The Tantalum keel was then made and placed as described by McNaught (Fig. 5-J) being careful not to have the keel too deep so that it might contact the posterior wall of the larynx and also making sure that good anchorage of the lateral flanges of the keel (Fig. 5-H) are made under the ribbon muscles.

7. The wound was closed with subcutaneous catgut sutures and skin closure made with Dermalon atraumatic sutures.

8. Early post-operatively the position of the keel was checked with P.A. and lateral X-rays.

9. The keel was removed eight weeks later after full corking and no dyspnea with heavy exercise and after inspection of the larynx revealed all inflammation within the larynx to have subsided.

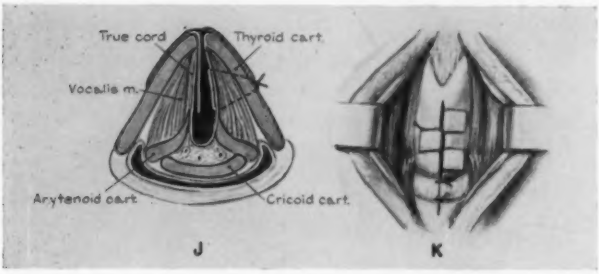

Fig. 5. (J) Placement of Tantalum Keel. (K) Anchoring keel with lateral flanges deep to ribbon muscles.

After removal of the keel her glottic chink (Fig. 6) appeared to be between 7 and 8 mm. width in its posterior aspect. This was a full normal anterior posterior aperature. She had no signs of dyspnea even on exertion. Her voice was adequate over the telephone, but was a whispered,

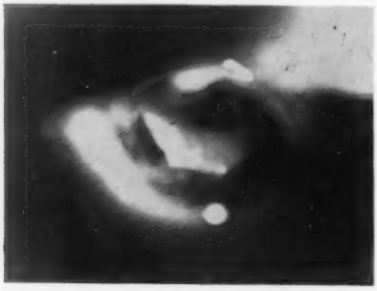

Fig. 6. Indirect View of Larynx six months post-op.

false cord type voice. It had little carrying powers, but still was adequate for all her needed functions as a home maker and permitted her to enter into any social conversation. Voice recording was made at this time. Seven weeks later patient returned to the hospital for plastic closure of her tracheal stoma.

DISCUSSION.

The technique described may be useful not only as in this case, where both cords had been operated upon, but also in cases in which an arytenoidectomy or lateralization procedure has been done on only one side and lateralization not accomplished.

I think in the latter cases it would be much better to attempt this type of technique, namely, submucosal cordectomy with lateral fixation of the mucosal flap and underlying soft tissues, as described, rather than attempt a corrective operation on the opposite side. This would leave one cord with good adductor powers and thus permit a better chance of stronger voice rehabilitation.

The question as to whether or not use should be made of the keel device should depend entirely on whether or not a web was present or whether any denuded surfaces opposed each other in such a manner as to favor web formation. If no such hazards were apparent it would not be needed and would thus save an additional operative step necessary for its removal.

The chief factor in gaining space is the submucosal removal of part of the true cord and the covering of this area with a viable flap on a hinge, also care not to incise beyond the true cord deep into muscle tissue, thus avoiding bleeding and hematoma formation. The method of applying the flap close to the lateral wall with the aid of Reverdin needle also helps to lateralize the underlying submucosal soft tissues, thus obtaining maximum gain in glottic space.

SUMMARY.

We have described a technique, taking advantage of the McNaught keel device, in which a plastic corrective procedure gained additional glottic space by the development of a viable mucosal flap on a hinge, with resection of the mid part of the true cord and lateralizing the mucosal flap and underlying soft structures, by the use of a suture placed with Reverdin needle, brought out and tied outside the thyroid ala cartilage.

This permitted successful decannulation of a patient who had had previous surgery on both cords for the relief of stenosis secondary to bilateral abductor paralysis.

We also suggest that this technique, with or without the use of a keel depending on indicated needs, may prove useful in those cases in which unilateral surgery for bilateral abductor paralysis had been done without gaining sufficient

lateralization to permit decannulation. By thus confining the corrective operative work to one side, you will preserve the full adductor powers of the opposite cord and have a better chance at stronger voice rehabilitation.

A Kodachrome Sound Track Film followed demonstrating the technique described, together with post-operative views of the patient's larynx eight months later.

Copies of this film may be obtained or rented through Sturgis-Grant Productions, 314 East 46th Street, New York 17, N. Y.

BIBLIOGRAPHY.

1. McNaught, Robert C.: "Surgical Correction of Anterior Web of the Larynx," The Laryngoscope, March, 1950, Vol. 60, page 264.

2. Moulonguet, A. *et al*: Rapports: Les Paralysies Bilaterales du larynx en adduction—Leur Traitement Chirurgical; Congres de 1951; *Societe Francaise d'Oto-Rhino-Laryngologie*, p. 39.

THE FORMATION OF A HEARING AND SPEECH CENTER.*

GORDON D. HOOPLE, M.D.
and
LOUIS M. DICARLO, Ph.D. (by invitation),
Syracuse, N. Y.

Even a cursory inspection of history reveals that from time immemorial the medical profession's basic objectives embraced: alleviating suffering, healing the sick, preventing disease, and, most important, improving the general welfare of a people. Because medicine is inextricably interwoven with a people's well being it has continued to progress through periods when the arts and sciences appeared sterile and dormant. Medicine by its very nature must function within the framework of the socio-economic parameter of a culture.[10] An evaluation of the status of the medical profession in a society often provides the sesame to the goals, ambitions, and aspiration levels of that society. The ancient Greeks crystallized this phenomenon in their profound apothem: "a sound mind in a sound body." Today, as in antiquity, medicine's primary devotion remains service to mankind. The illusion that hearing and speech impairments are single chronic entities is slowly evaporating. The tempo and complexity of modern civilization requires continuous readjustments in human relationships. Extensive accumulation and diversification of knowledge in general, gigantic advances in medicine, new and more refined techniques for diagnosis, more efficacious methods of treatment of existing pathologies have precipitated our high level specialization. Today most experts agree that any total rehabilitation program requires the team approach. There is also growing awareness that the care and even clearing up of the physical disability does not necessarily guarantee

*Read at the 57th Annual Meeting of the American Laryngological, Rhinological and Otological Society, New Orleans, La., April 28, 1953.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, May 4, 1953.

resolution of the handicap; some physical disabilities are irreversible. In these instances, the rehabilitation program should function to arrest and minimize the psychological disturbances, educational retardation, and the vocational maladjustments that may result from the physical disability through improper mental hygiene handling of the handicap. Of all the impairments which afflict our population, few corrode the personality with greater unhappiness, cause more real tragedy, and result in more maladjustments, than hearing and speech impairments; for, unless the hearing or the speech handicapped individual is guided into a pattern of educational, emotional, and vocational security, bewilderment, intellectual disillusionment, and emotional frustration may conspire towards the development of an unhealthy basic personality structure.[4,5] How hearing and speech impairments warp, twist, and influence behavior has been recognized by a few for some time, but it has been only recently that the full force of the impact of these impairments have become clarified. World War II provided a great impetus towards an understanding of the severity of these impairments and the necessity for the development of an adequate rehabilitation program. The army and navy rehabilitation programs for hearing and speech casualties provided a beginning conceptual model for some of the present university clinics.

Walsh[11] has discussed the tri-function role of a University Hearing and Speech Center: teaching, research, and service. We would also include in his team the pediatrician, dentist, and the social worker; but the University Hearing and Speech Centers serve only a small portion of the population who require a total program of rehabilitation. Some interested otologists have equipped their offices with equipment that permits them to obtain additional information in order that they may provide the initial counseling and orientation for their patients. Such procedures can be quite lengthy and sometimes not feasible at one conference. Some communities meet the demands for this service by the establishment of hospital centers where the different specialities contribute to the overall program. Another type of center

develops as a community project. Such a hearing and speech center often represents the ideal of a group or even one doctor to serve the hearing and speech defective individual in a greater capacity through the coordinated efforts of a number of specialties. Hill[7] has described this organization in some detail. Unfortunately, there exists too few of these centers. Our guess, based on our own files, indicates that all the centers in the country today probably serve less than twenty-five per cent of the children and adults who require rehabilitation. One of the reasons for the existence of too few centers may be attributed to the delusion that such a center must have extensive facilities and personnel for its operation.

We are quite concerned with the general lack of such facilities. We are strongly convinced that the care and the rehabilitation of hearing and speech impaired individuals is a community responsibility. The formation of a community hearing and speech center would appear to us to be a social imperative. A hearing and speech center is not a purely material entity. The formation of a hearing and speech center embraces the thinking, the ideals, and a way of living in our democratic social structure. The formation of a community hearing and speech center embraces primary and secondary objectives, but the basic philosophy remains one of preserving each individual's integrity and also the provision of an equality of opportunity for physical, mental, social, and economic development.

For the hearing and speech handicapped individual, the total rehabilitation program will be based on the findings of medicine. Consequently, the key to the process is the otologist, or otolaryngologist. He must be the key member in the formation of a hearing and speech center. If he fails to understand the problems related to the handicap, then the rehabilitation process breaks down. Medicine can no longer be interested only in the ailments of the body but must concern itself with the total individual. When the

general health problems have been clarified, the otologist must assume the leadership for the direction and operation of the center.

Housing becomes the next primary consideration. Where the center will be housed depends upon the interest of the medical profession and the community resources available. A hospital may provide a room or two at specific times to be used for that purpose. Where this is not feasible, a school system might provide space for such housing. In many communities where the school is overcrowded, the basement of a church or any community center may be utilized.

The third primary consideration involves equipment. It is our feeling that this consideration has prevented the formation of many community hearing and speech centers because of the unrealistic goals that the founders of such centers set up for themselves. In the beginning, such a center could function with one commercial audiometer which meets the standards of the Council on Physical Medicine and Rehabilitation of the American Medical Association. Another piece of equipment would be either an inexpensive recording machine or a tape recorder. Most communities have a number of service clubs whose help could be solicited in the procurement of this equipment. In our experience, we have found the service clubs to be most cooperative and willing to purchase such equipment if the financial outlay were modest and realistic. Additional equipment would include an auditory training unit, since we consider auditory training to be one of the most important educational differentials in the development and refinement of the interpretative responses. Bordley[1] and Lewis[2] have made significant contributions in the evaluation of early auditory training for children with profound hearing losses. Rapid strides in electronic equipment, especially research dealing with improvement in amplifying devices aimed at producing significant gains in audiometric curves and speech reception thresholds, have permitted us to help more and more of these children with seriously impaired hearing. There are a number of commercial auditory training units on the

market, but in almost every community, there are interested people who have electronic background and training. It has been our experience that they are willing to contribute time and labor in helping to construct such a unit. For example; Fig. 1 is a unit which we have used with our deaf and hard of hearing children that was constructed for less

Fig. 1. An amplification unit with six outlets, flat response curve up to 6000 cycles, maximum output of 120 decibels including separate play-back and talk-back provisions.

than two hundred dollars. It has six outlets, a flat curve response up to 6000 cycles with a maximum output of 120 decibels including provisions for a separate play back and talk-back unit. If the Center cannot afford an auditory training unit of this kind, auditory training could be conducted with a simple table model hearing aid which could be constructed at a cost of about thirty-five to forty dollars. The cost of equipment should not prove a deterring factor in the formation of a hearing and speech center. We should think in terms of service and not facilities. A sound treated room for the conducting of tests and the evaluation of

hearing aids would also be necessary. Fig. 2 is a simple booth that would serve as a room within a room constructed of fiber and plastic board with studs and a layer of rock-wool between the inner and outer wall which would cost

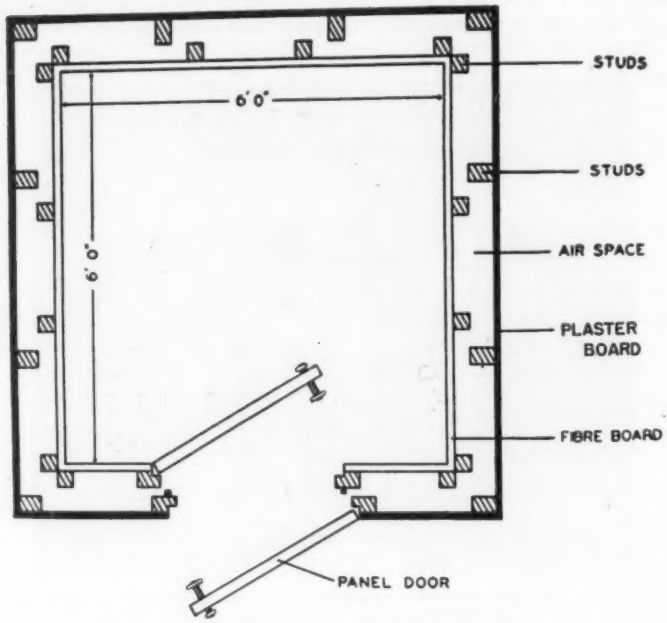

Fig. 2. Simple sound treated booth for hearing testing.

about three hundred dollars. If this cubicle could be placed in a room already within a room, there would be enough sound attenuation that would make it perfectly practical. This could then be used as a testing room. Fig. 3 is a picture of one of our rooms which cost about sixty dollars to sound treat. We found this quite satisfactory in the performance of tests.

Regarding the evaluation of hearing aids, Carhart[2] has suggested certain straightforward procedures. He has also suggested monitored live voice as a test for auditory acuity.[3]

An individual who understands the acoustic problems involved and the skills to be measured by the different tests, could borrow equipment or use normals to standarize the noise levels and the other thresholds in the room, but for a

Fig. 3. Sound treated room for hearing testing.

reasonable financial outlay, a play-back could be constructed which would be fed into the booth we have discussed and better controlled types of testing situations could be setup. A hearing aid evaluation room, such as Fig. 4, could be setup for about seven hundred dollars, but as we have already indicated, the lack of such a room should not be an obstacle to the formation of a hearing and speech center. Speech reading and socialization could be provided for concomitantly with auditory training in a relatively quiet room without the benefit of sound treatment. In fact, sometimes this situation is preferrable in order to provide degrees of listening difficulties which the individual experiences in a competitive noisy world.

In addition to the medical staff, the next important member of the team would be the audiologist. He may be the

cnly paid professional member of the team. Dr. Walsh[11] and Dr. Wishart[12] have considered his function in a hearing and speech center, but the otologist, because of the nature of the problems and because of this interest in the ultimate welfare of the handicapped individuals must assume primary

Fig. 4. Sound treated room for hearing aid evaluation.

leadership in the formation of such a hearing and speech center. The audiologist becomes the educational director. His function lies in the educational and vocational rehabilitation of these people. He is another member of the team. Provision of salary becomes a problem to most communities, but in most states such educational services may be supported by the state. In addition, his salary may be subsidized by a foundation or an organization such as the National Association for Crippled Children and Adults. A hearing and speech center requires his services, but there are many ways in which financing his services may be made available to the community. In some communities, the school system provides this service on a part-time basis. The services of

the other specialties such as the social worker and a teacher of a pre-school group may be procured through the aid of a voluntary organization. It would be both the otologist's and the audiologist's function to train these people in understanding the many ramifications of hearing and speech impairments. It would be the responsibility of the audiologist and speech pathologist to provide the parents with the necessary training material for hearing, speech and language development. It would also be his job to provide a flexible plan which would include the cooperation of the child's classroom teacher. With respect to the children, the crux of the problem is the child's regular teacher. If she is poorly oriented, she sees the child as a source of extra work. Unless the teacher becomes sematically oriented, she can frustrate any plan for a program. The program of such a center would culminate not in the provision of one specific skill but in a number of skills. The stage must be set for transfer in order that they may not be only isolated values. With the team approach, the possibility of such transfer could be continuously reinforced.

The secondary objective of a hearing and speech center involves the acquisition of more elaborate equipment, housing facilities and the provision of a greater number of services. These will come as the Center is already established. In the formation of a community hearing and speech center, minimum equipment, minimum personnel, space and an actual working unit must precede more ambitious ideals. The primary function of a hearing and speech center would be the prevention of hearing and speech impairments and arresting and minimizing the handicap. Kinney[8] indicates that a good deal of auditory impairment in children could be prevented by early detection and proper treatment. Where the handicap is irreversible, the function of the hearing and speech center becomes one of providing medical, educational, psychological and vocational services which will make for maximum healthy adjustment. In some cases, such a center may not be able to provide a complete array of specialties for the resolvement of certain problems. In these perplexing situations, the

center might have recourse to a university center, but for the majority of its people, the community hearing and speech center would provide a wonderful array of services and prevent the frustration and discouragement that these handicapped individuals experience when left to themselves to find solution to their problems.

In summary, we would reiterate our conviction that the formation of a hearing and speech center depends primarily on the willingness of the otologist to roll up his sleeves and go to work. If he is willing, the housing, equipment, and personnel will follow. The cost of administering such a hearing conservation program will be negligible in terms of a total budget, but in terms of salvaging human self esteem, through realistic self appraisal of both the restrictions and possibilities; alleviating human suffering and conflict and providing for physical, intellectual, emotional, and social civic growth there can be no greater remuneration. The provision of a rehabilitation program would be a noble task worthy of the traditions in our great democracy.

REFERENCES.

1. BORDLEY, JOHN E. The Problem of the Preschool Child. THE LARYNGOSCOPE, LXII No. 5, (May 1952) 514-520.

2. CARHART, RAYMOND. Hearing Aid Selections by University Clinics. *Journal of Hearing and Speech Disorders*, Vol. 15, No. 2 (June 1950) 106-113.

3. CARHART, RAYMOND. Monitored Live Voice as a Test of Auditory Acuity. *Journal of Acoustical Society of America.* Vol. 17, No. 4, (April 1946) 339-349.

4. DICARLO, LOUIS M. An Educational Program for Children with Impaired Hearing. *Elementary School Journal.* XLIX (November 1948) 160-167.

5. DICARLO, LOUIS M. Mental Hygiene Approach to Hearing and Deafness. *Quarterly Journal of Child Behavior.* III No. 3, (July 1951) 318-322.

6. HARDY, WILLIAM G. Special Techniques for Diagnosis and Treatment of Psychogenic Deafness. *Annals of Otology, Rhinology, and Laryngology.* Vol. 57, No. 1, (March 1948), 65-91.

7. HILL, FREDERICK T. and KOONS, ELIZABETH D. Essentials for Auditory Rehabilitation. *Annals of Otology, Rhinology, and Laryngology.* Vol. 61, No. 3, (September 1952), 751-756.

8. KINNEY, CHARLES E. Hearing Impairments in Children. THE LARYNGOSCOPE. LXIII No. 3, (March 1953) 220-226.

9. LEWIS, DONALD K. The Rehabilitation of the Preschool Deaf Child. THE LARYNGOSCOPE. LX No. 6, (June 1950) 564-576.

10. LINTON, RALPH. The Science of Man in the World Crisis. New York: Columbia University Press, 1945.

11. WALSH, THEODORE E. Audiology, Its Relationship to Otology. *Trans. Amer. Acad. of Ophthal. and Otolaryngol.* Vol. 56, No. 1, (January-February 1952) 78-80.

12. WISHART, D. E. S. A Critique of Present Day Methods of Hearing Testing. THE LARYNGOSCOPE. LXII No. 10, (October 1952) 1045-1068.

MASKING AND FATIGUE EFFECTS OF WHITE NOISE IN CONNECTION WITH SPEECH TESTS IN OTOSCLEROTIC SUBJECTS.*

CARLO SAMBATARO, M.D., and GIULIO PESTALOZZA, M.D.,
Milan, Italy.

In a preceding study we examined the behavior of threshold shift produced by white noise in speech tests with logatomes on persons with normal hearing at progressive intensity levels above threshold. These researches were carried out in a free field, using loudspeakers.

The threshold shift was small for low levels of white noise, becoming larger as the white noise was increased in volume. The phenomenon is illustrated by the full line curve in Fig. 3.

A further observation made in our previous work showed that when a subject with normal hearing is exposed for 15 minutes to white noise at a high intensity level (60 to 80 db above threshold) the articulation curve for logatomes improves by approximately 5 db along its entire length.

In order to explain this phenomenon, we advanced the following three hypotheses: *1.* reduction of the muscular tone of the middle ear muscles, particularly of the stapedium, due to the fatigue of these muscles caused by the prolonged stimulation to which they have been subjected. This hypothesis should be linked to the phenomenon of "hyperacusis dolorosa" observed in many cases where facial nerve palsy produces consequent palsy of the stapedium; *2.* accumulation of chemical mediators of the nervous system, caused by the sound stimulus, which facilitates the transmission of the sound current through the interneuronic synapsis; *3.* psychic effect due

*From the Ear, Nose and Throat Clinic, University of Milan, Italy.
Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Aug. 30, 1952.

to the absolute and sudden silence following a condition of high intensity prolonged noise, causing the subject better to perceive speech, both subjectively and objectively.

In our previous work we stated that we proposed to study the threshold shift in cases of otosclerosis and to attempt to determine which of our three hypotheses, as explanation of the above mentioned phenomenon, was correct. In order to do this we intended repeating the previous tests carried out on subjects with normal hearing, on otosclerotic subjects, where the ankylosis of the stapes eliminates in practice the contraction of the stapedius muscle.

Our present experiments refer to six subjects with otosclerosis in the second stage of Shambaugh with good bone conduction preservation. Pure tone audiograms and complementary acoustic tests (Gellé, Bing, bone masking) are shown in Fig. 1.

Testing conditions and methods used were identical with those used when studying subjects with normal hearing and are as follows: The subject was made to enter a silent cabin and to sit down one meter from the two loudspeakers placed one above the other (one emitted white noise while the other transmitted logatomes); the examiner, outside the silent cabin, was thus able to carry out the articulation curve for logatomes in silent conditions; by switching on the white noise generator, he was then able to repeat the articulation curves at the following fixed intensity levels of white noise: 20, 40, 60 db above threshold. Having finished this first part of the test, and under identical conditions as previously, the subject being examined was required to listen to the white noise at 60 db above threshold intensity level for 15 minutes with the intention of introducing a state of fatigue, after which the articulation curve was repeated immediately after the white noise had been switched off. The above research method was critically analyzed in our preceding work.

We now consider the results of the first part of our research concerning threshold shift. Comparing the curves

showing threshold shift in the normal ear with that of the otosclerotic ear, bearing in mind that the values shown on the graph refer exclusively to intensity levels above the threshold of the subject being examined as reduced to zero (see Figs. 2 and 3), it is noted that for low intensity levels of

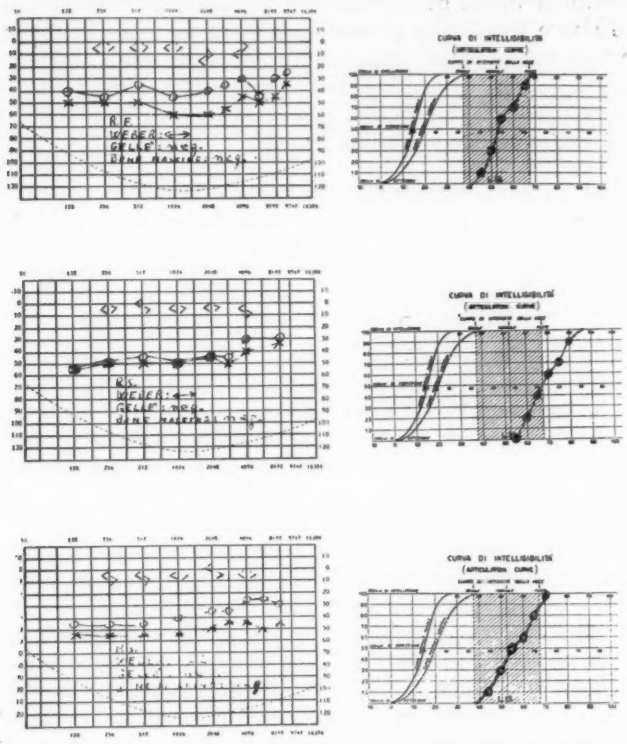

Fig. 1. Audiograms and articulation curves of the otosclerotic subjects mentioned in the text.

white noise, the shift is far lower in the case of otosclerosis than in the normal (7 db for normal, only 3.2 db in the case of otosclerosis); for higher intensity values of white noise above threshold, the two curves practically coincide.

This means that the masking effect of low volume white noise (20 db above threshold) on logatomes perception is lower in the otosclerotic than in the normal, while for higher intensity levels (40 to 60 db above threshold) the otosclerotic ear behaves as does the normal.

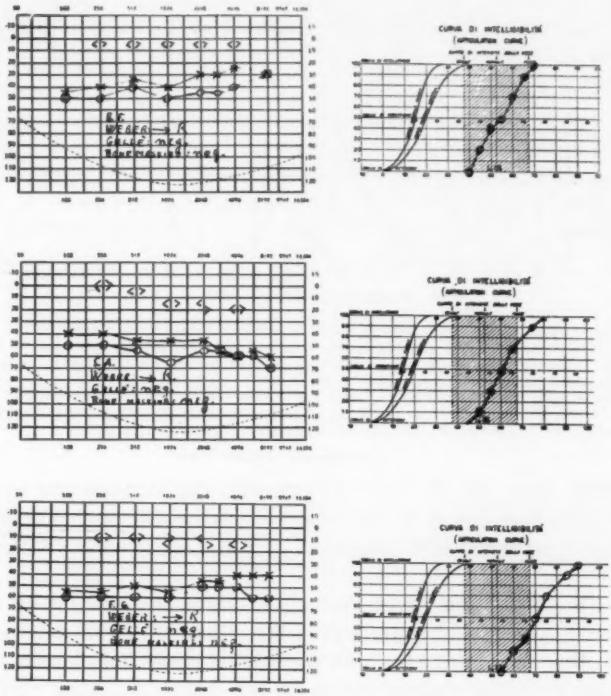

Fig. 1. Audiograms and articulation curves of the otosclerotic subjects mentioned in the text.

Our results concur with those of Azzi, which refer to threshold shift for pure tones when a masking noise with saw-tooth wave form at 20 db level is used. The above named author did not repeat his test with masking at higher levels, and it is thus impossible for us to compare our complete results.

The second part of our test enables us to demonstrate that when an otosclerotic subject is exposed to white noise for 15 minutes at the maximum intensity level permitted by his

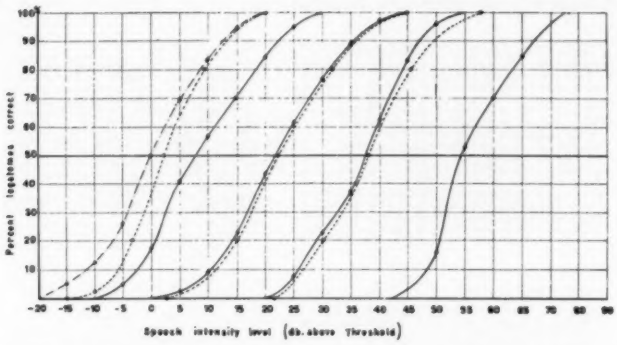

Fig. 2. The first curve (shown with alternate dots and dashes) represents the mean value of the curves of the six subjects examined reduced to zero level. The curves shown as a series of dashes represent the mean value of threshold shift in the presence of white noise at intensities of 20, 40, 60 db above the individual threshold of each otosclerotic subject examined. The continuous line curves represent the same phenomenon with normal subjects at intensity values of 20, 40, 60, 80 db above normal threshold.

hearing loss (60 db above threshold), the articulation curve does not improve as in the case with normal subject; in fact, it remains practically unvaried (see Fig. 3).

This affirmation gives undoubted value to the first of the three hypotheses mentioned at the start of these notes; in fact, the only point at which the second stage otosclerotic ear varies with the normal ear is in the mobility of the stapes, which is fixed in the former and mobile in the latter. It is this rigidity which eliminates any effects caused by contraction or relaxation of the stapedius muscle.

As the threshold in the case of otosclerosis is not improved, it is probable that the phenomenon noticed in normal subjects is determined by the fatigue condition of the stapedius caused by prolonged and intense sound stimulation.

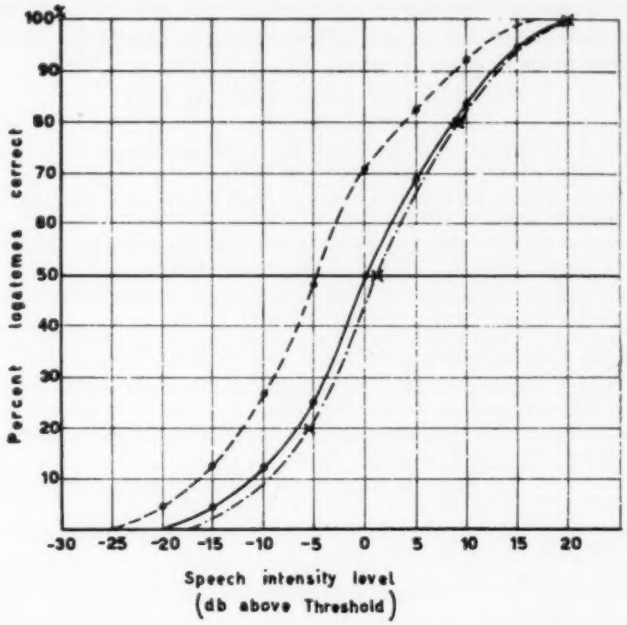

Fig. 3. The continuous line represents a normal articulation curve. The interrupted dash line represents the articulation curve obtained after the normal subject has been exposed to white noise at 60 db intensity level above threshold. The line shown with alternate dots and dashes represents the same phenomenon for the otosclerotic subject at the same relative intensity values above threshold.

SUMMARY.

The authors, referring to their recent work regarding threshold shift caused by white noise on normal subjects report on the results obtained with otosclerotic subjects by identical research methods. The threshold shift in cases of otosclerosis is lower for low intensities of white noise and similar to normal for medium and high intensities.

The improvement in threshold after exposure to white noise for 15 minutes always encountered in the normal sub-

ject, is not encountered in otosclerosis; this gives value to the hypothesis that the fatigue of the stapedius muscle is a cause of this phenomenon.

BIBLIOGRAPHY.

AZZI, A.: Prove di acumetria vocale per la lingua italiana. *Arch. It. di Otol., Rinol. e Laring.*, 61, Supp. 5, 45-85, 1950.

AZZI, A., e INVERNIZZI, M.: La deriva di soglia. *Arch. It. di Otol., Rinol. e Laring.*, 61, Supp. 5, 154-180, 1950.

EGAN, J. P.: The Effect of Noise in One Ear Upon the Loudness of Speech in the Other Ear. *Jour. A. S. A.*, 20:1:58, 1949.

HARRIS, J. D.: The Effect of Signal Noise Levels on Pitch Discrimination in masking. *Jour. A. S. A.*, 19:1:293, 1947.

HARRIS, J. D.: The Effect of Noise Sensation Level Upon Pitch Discrimination in a Continuous Thermal Noise. *Jour. A. S. A.*, 19:4:733, 1947.

HAWKINS, J., and STEVENS, S. S.: Masking of Pure Tone and Speech by White Noise. *Jour. A. S. A.*, 20:1:6-13, 1947.

LICKLIDER, J. C. R., and GOFFORD, S. J.: Effect of Noise on Radio Communication. *Jour. A. S. A.*, 19:4:743, 1947.

POLLACK, I.: The Effect of White Noise on the Loudness of Speech of Assigned Average Level. *Jour. A. S. A.*, 21:3:255, 1949.

SALTZMANN, M., and ERSNER: Masking as an Aid in Audiometry. THE LARYNGOSCOPE, 58:10:1127-1132, 1948.

SAMBATARO, C., & PESTALOZZA, G.: Masking and Fatigue Effect of White Noise in Connection with Speech Tests. THE LARYNGOSCOPE, 62:11:1197-1204, NOV., 1952.

EVALUATION OF A NEW SPRAY DEVICE FOR INTRANASAL MEDICATION.

D. G. ORNSTON, M.D.,
Philadelphia, Pa.

This is a report on a "gadget." This gadget is a small polyethylene bottle which, when held to the nose and squeezed, releases a fine cloud of medication into the nasal cavities. The novelty of this container has obvious commercial appeal, and in the last year about a dozen of them have been marketed as a means of administering many well known nasal medications. Ostensibly, this plastic bottle should be as effective as an ordinary nasal spray, but it occurred to me that there was a good chance that it could not release enough suspended droplets of the medication, or release them fast enough, to insure adequate coverage of the nasal membranes.

The nasal tract is a very involved structure, and medication must be carefully administered for the drug to reach those areas where it is most needed. The two most popular and effective forms for self-administered nasal medication are, of course, the spray and the drop; but the large bulb and long tip of even the smallest standard atomizer make the public use of it a rather disconcertingly conspicuous exhibition, and the proper administration of nose drops depends on postural attitudes which some patients find awkward and at times impossible to assume. The gadget may supply a long needed answer to the vexing problem of self-administration of intranasal therapy — if it works. It was to find out if it does work (*i.e.*, if it is mechanically efficient) that the following study was carried out.

The spray bottle was evaluated in three ways: first, by observing the nasal coverage of sulfathiazole crystals obtained by its use; second, by comparing the results of spray and drop

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, March 27, 1953.

application of the same medication in 49 patients; and last, by making X-ray studies of a radio-opaque medium administered by the drop and spray bottle methods.

The first test was a very simple one. Since sufathiazole crystals can easily be seen in the nasal cavity, a nasal medication[1] was selected which is available in the spray bottle and which combines sulfathiazole with a vasoconstrictor. Observations were made in about a dozen patients immediately after they had received this preparation. Repeated examinations disclosed that the area of the mucosa reached by the medication depended, not surprisingly, on the patency of the common meatus. The first inspiration of the sprayed material did not usually result in maximum coverage but did shrink the turbinates through the action of Paredrine, the vasoconstrictor contained in this medication. When another application was given shortly after the first, the sulfathiazole crystals reached the olfactory fissure. These results seemed to indicate that, in all probability, the spray bottle might be effective in treating patients whose nasal congestion was no more than moderately severe. Accordingly, the following clinical study was undertaken.

†The medication chosen for this study was an isotonic aqueous solution containing two antibiotics (gramicidin and polymyxin), an antihistamine (thenylpyramine) and a vasoconstrictor (Paredrine) which is available in both the solution* and spray bottle**.

Forty-nine patients who complained of nasal congestion were chosen for this study. Nasopharyngoscopic examinations were made, and each condition was diagnosed as either infectious or allergenic or a combination of both. No patient was chosen who had a nasal congestion severe enough to prevent the drops or spray from reaching beyond the inferior turbinate. Since all were concurrently treated—some systemically with penicillin, others by displacement therapy, bed rest, etc. —no attempt was made to determine the effect of the nasal

†P.S.S. Spraypak. This and the other drug used in this study (*Drilitol, **Drilitol Spraypak) were supplied by the Smith, Kline and French Laboratories.

medication on the course of the infection. The present study, then, was designed to test the efficiency of the dispenser, not the efficacy of the drug.

In the first part of the experiment, 5 drops of medication were instilled in one nostril; the amount of medication released by three squeezes of the container (about 3 drops) was sprayed into the other. The time necessary for objective evidence of lessened congestion was noted, and the patients' comments recorded.

The amount of time it took either form to produce objective evidence of lessened congestion did not differ significantly in 26 patients. The spray had a slightly quicker effect in 15 patients. The drops had a slightly quicker effect in eight patients.

Most patients claimed an almost immediate, beneficial shrinking effect from the medication although there was no perceptible increase of nasal patency for about six minutes. It is possible that the mere application of treatment created a feeling of confidence and increased well being, or that the medication resulted in some slight shrinkage which could be felt instantly by the patient but which was not great enough to be seen by me.

Each patient was then given the plastic spray bottle for his personal use. On the next visit the patients' comments were again noted. An evaluation of the coverage provided by the spray (based on the degree of relief experienced by the patients) gave the following results:

Degree of Coverage	Number of Patients
Excellent	1
Good	31
Fair	11
Poor	6

The duration of relief achieved by the spray (about 2 hours) was quite satisfactory. Eight patients said that the spray treatment was not so effective as previous treatment with nose drops, and claimed that it was irritating, increased nasal congestion, or was simply "not so good as" drops. Twenty-two

patients were enthusiastic about the spray. Two of these (a college student and a bus driver) were particularly impressed by its convenience and the inconspicuous way in which it administered the medication. Other patients remarked that the spray "didn't make me gag the way drops always do," soothed the throat, relieved cough and got rid of pressure in the frontal sinus. Nineteen patients thought the spray was effective but no better than nose drops.

The fact that many patients preferred the spray to drops is not considered as too significant since, in all probability, the novelty of the gadget contributed a good deal to its popularity; however, the fact that most patients received satisfactory relief while using the spray bottle indicated that it provided adequate coverage.

It was then decided to try to obtain X-ray evidence to find out precisely how adequate this coverage was. X-rays, it was felt, would show the efficiency of both the spray and the drop methods by revealing which procedure produced the best filling and reached the highest level of the nose.

The patient chosen was a young woman just recovering from an acute ethmoiditis. A 30 per cent solution of Uroken (a radio-opaque medium with approximately the same viscosity as the medication) was sprayed, and later dropped, into each nostril. Excerpts from the X-ray report follow:[2]

> The opaque material did not reach above the level of the top of the inferior turbinates when sprayed into the nose until the membranes were pre-shrunk. At the time of the last examination the sprayed Uroken was seen at the middle turbinate level.
>
> The drops found their way into the ethmoid cells both anteriorly and posteriorly at the time of the examinations done 12/17 and 12/22/52. More of the opaque material is seen in the ethmoids after the instillation done 12/22/52 than was present 12/17/52.
>
> Conclusions: It is possible to visualize sprayed opaque material as high as the middle turbinate level when the mucous membrane of the nose is shrunken by previous instillation of a proper solution. Otherwise it does not get above the level of the inferior turbinates. Drops reach the roof of the nose and the anterior and posterior ethmoid cells superiorly when instilled in the Proetz position.

How well the drops reached the roof of the nose and into the anterior and posterior ethmoid cells came as a revelation

to me. It was surprising to see how well some of the ethmoidal cells could be filled simply by applying the drops while the patient's head was upside down.

The results of these X-rays* indicate that while the plastic spray bottle is not so efficient a means of administering medication as properly applied nose drops, it does supply better than adequate coverage. It is also apparent that the patient, in order to obtain the best coverage from the spray, should use it once to open the inferior part of the common meatus, *then again a few minutes later,* to shrink the turbinates and provide adequate sinus drainage.

Judging from the results of each of these three experiments, it would seem that the plastic spray bottle is a mechanically efficient device which is of value in the treatment of intranasal infection. Although not so efficient as properly administered nose drops, it is probably the treatment of choice for those patients who find it impossible or inconvenient to use the drops properly.

SUMMARY AND CONCLUSION.

1. The mechanical efficiency of the plastic spray bottle — a new aerosol form for the administration of intranasal medication — was tested in three ways: by observing the nasal coverage of sulfathiazole crystals obtained by its use; by comparing the results of spray and drop application of the same medication in 49 patients; and by making X-ray studies of a radio-opaque medium administered by the drop and spray bottle method.

2. The first test showed that the effectiveness of the spray depended upon the patency of the common meatus. If the turbinates were first shrunken by one application of the spray, another application given shortly afterward enabled the medication to reach the olfactory fissure.

*These X-rays were taken at the Germantown Hospital in Philadelphia. Appreciation is extended to Dr. Barton R. Young and Dr. Robert L. Scanlon for their kind cooperation.

3. The clinical study of 49 patients given — in addition to other therapy — treatment by both spray and drop methods revealed no important observable difference in the effectiveness of the two methods. The spray bottle seemed to provide adequate coverage.

4. X-ray examinations showed that the spray bottle, while not providing so good a coverage as properly applied drops, did administer the medication effectively.

5. It was concluded that the plastic spray bottle is a mechanically efficient device and a valuable adjunct to the treatment of intranasal infections.

THE STORY OF THE "MAGIC FLUID"

Of all the magic gadgets in the past, including the famed lamp of Aladdin, there are none that compare with blood . . . the "Magic Fluid." Despite the wonderful gifts that these magic-makers were reported to bestow, none could boast of giving life, for only the wonder-maker blood can restore ebbing life.

Stone-age man knew about the importance of blood for he wrote about it with crude carvings on the stone walls in the depths of his caves. On every page of history there is some notation about blood. Man has never lost his interest in blood from the first dawn of light in the world. Progress of knowledge through the centuries was tragically slow. Not until 1942 did large scale use of blood become a reality. Step-by-step through World War II blood application techniques were proven through thousands upon thousands of blood transfusions to save lives of wounded fighting men.

Another major advance was the discovery that this magic fluid contained Gamma Globulin for use in fighting disease. Gamma Globulin is now being made in millions of doses to prevent measles, infectious hepatitis and the paralytic effects of polio. Constant intensive research promises even far greater discoveries in the struggle against disease and death.

You are lucky to be living in the age that discovered the magic of blood. It is a gift to help make you live longer . . . better. The "Magic Fluid" supply depends on you. You can help keep the fountain of blood flowing . . . with all of it's life-saving magic. Call your local Red Cross Community or Armed Forces Blood Donor Center today to schedule your contribution to the supply of this "Magic Fluid."

MISSISSIPPI VALLEY MEDICAL SOCIETY

Arthur H. Keeney, M.S., M.D., Clinical Instructor in Ophthalmology, University of Louisville, is the winner of the 13th Annual Essay Contest, Mississippi Valley Medical Society, "for the best unpublished essay on a subject of practical and applicable value to the general practioner of medicine." Dr. Keeney's paper is entitled "Grass Roots in the Prevention of Blindness." Second prize goes to Dr. Louis T. Palumbo, Chief, Surgical Service, Veterans Administration Center, Des Moines, Ia., for his paper "Physiological Changes of the Upper Gastrointestinal Tract Following Combined Upper Gastrectomy and Vagus Resection." There was a tie for Third Prize—Dr. A. Henry Clagett, Jr., of the Veterans Administration Hospital, Wilmington, Del., for his paper "Some Common Complications of Myocardial Infarction" and Dr. Walter M. Block, of Cedar Rapids, Iowa, for his paper "Infant Feeding with Homogenized Milk." Dr. Keeney will receive a cash award, a gold medal, a certificate of award, and will present his essay at the 18th Annual Meeting, Mississippi Valley Medical Society, Springfield, Ill., Sept. 24. His paper will appear in the January 1954 issue of the Mississippi Valley Medical Journal (Quincy, Ill.).

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

August 1, 1953.

Audicon Models 400, 415, 530 and 615.
Manufacturer: National Earphone Co., Inc., 20-22 Shipman St., Newark 2, N. J.

Auditone Models 11 and 15.
Manufacturer: Audio Co. of America, 5305 N. Sixth St., Phoenix, Ariz.

Audivox Model Super 67 and 70.
Manufacturer: Audivox, Inc., 259 W. 14th St., New York 11, N. Y.

Aurex Models L and M.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Symphonette; Beltone Mono-Pac Model M; Mono-Pac Model "Lyric"; Mono-Pac Model "Rhapsody."
Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Cleartone Model 500; Model 700; Cleartone Regency Model.
Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Dahlberg Model D-1; Dahlberg Junior Model D-2; Dahlberg Model D-3 Tru-Sonic; Dahlberg Model D-4 Tru-Sonic.
Manufacturer: The Dahlberg Co., Golden Valley, Minneapolis 22, Minn.

Dysonic Model 1.
Manufacturer: Dynamic Hearing Aids, 149 Church St., New York 7, N. Y.

Fortiphone Models 19-LR; 20A; 21-C and 22.
Manufacturer: Fortiphone Limited, Fortiphone House, 247 Regent St., London W. 1, England.
Distributor: Anton Heilman, 75 Madison Ave., New York 16, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Goldentone Models 25, 69 and 97.
Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40th St., Minneapolis 8, Minn.
Distributor: Goldentone Corp., 708 W. 40th St., Minneapolis 8, Minn.

Maico UE-Atomeer; Maico Quiet Ear Models G and H; Maico Model J; Maico Top Secret Model L.
Manufacturer: Maico Co., Inc., 21 North Third St., Minneapolis, Minn.

Mears (Crystal and Magnetic) Aurophone Model 200.
Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 303; Micronic Model "Mercury"; Micronic Star Model.
Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Microtone Classic Model T9; Microtone Model T10; Microtone Model T612; Microtone Model 45.
Manufacturer: Microtone Co., Ford Parkway on the Mississippi, St. Paul, Minn.; Minneapolis 9, Minn.

National Cub Model C; National Cub Model D (Duplex); National Standard Model T; National Star Model S; National Ultrathin Model 504; National Vanity Model 506.
Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Normatone Model C.
Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40 St., Minneapolis, Minn.
Distributor: Normatone Hearing Aid Co., 22 East 7th St., St. Paul (1), Minn.

Otarion Model E-4; Otarion Models F-1, F-2 and F-3; Otarion Model G-2; Otarion Model G-3; Otarion Model H-1; Custom 5.
Manufacturer: Otarion Hearing Aids, 4757 N. Ravenwood, Chicago 40, Ill.

Paravox Model D, "Top-Twin-Tone"; Model J (Tiny-Mite); Paravox Model Y (YM, YC and YC-7) (Veri-Small).
Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear All Magnetic Model 55; Radioear Model 62 Starlet; Model 72; Model 82 (Zephyr).

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Distributor: Radioear Corp.

Silvertone Model J-92; Silvertone Model P-15.

Manufacturer: W. E. Johnson Mfg. Co., 708 W. 40th St., Minneapolis, Minn.

Distributor: Sears, Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.

Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925; Sonotone Model 940; Sonotone Model 966.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.

Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Televox Model E.

Manufacturer: Televox Mfg. Co., 1307 Sansom St., Philadelphia 7, Pa.

Telex Model 97; Telex Model 99; Telex Model 200; Telex Model 300B; Telex Model 400; Telex Model 500; Telex Model 952; Telex Model 953; Telex Model 1700.

Manufacturer: Telex, Inc., Telex Park, St. Paul 1, Minn.

Tonamic Model 50.

Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster; Model Cameo.

Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill.

Unex Midget Model 95; Unex Midget Model 110; Unex Models 200 and 230.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Models J and J-2.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Zenith Miniature 75; Zenith Model Royal; Zenith Model Super Royal; Zenith "Regent."
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS.

Ambco Hearing Amplifier (Table Model).
Manufacturer: A. M. Brooks Co., 64 S. Bonnie Brae St., Los Angeles 5, Calif.

Aurex (Semi-Portable).
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago 10, Ill.

Precision Table Hearing Aid.
Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

AMERICAN OTOLOGICAL SOCIETY.

President: Dr. Frederick T. Hill, Professional Bldg., Waterville, Me.
Vice-President: Dr. D. E. Staunton Wishart, 170 St. George St., Toronto 5, Ontario, Canada.
Secretary: Dr. John R. Lindsay, 950 E. 59th St., Chicago 37, Ill.
Editor-Librarian: Dr. Henry L. Williams, Mayo Clinic, Rochester, Minn.
Meeting: Statler Hotel, Boston, Mass., May 16-17, 1954.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Gordon F. Harkness, Davenport, Iowa.
First Vice-President: Claude C. Cody, Houston, Tex.
Second Vice-President: Daniel S. Cunning, New York, N. Y.
Secretary: Harry P. Schenck, Philadelphia, Pa.
Treasurer: Fred W. Dixon, Cleveland, Ohio.
Meeting: Statler Hotel, Boston, Mass., May 20-21, 1954.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. LeRoy A. Schall, 243 Charles St., Boston, Mass.
President-Elect: Dr. Kenneth M. Day, 121 University Pl., Pittsburgh, Pa.
Secretary: Dr. C. Stewart Nash, 277 Alexander St., Rochester, N. Y.
Meeting: Statler Hotel, Boston, Mass., May, 1954.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

Chairman: Dr. Carl H. McCaskey, 608 Guaranty Bldg., Indianapolis, Ind.
Vice-Chairman: Dr. Fred W. Dixon, Rose Bldg., Cleveland, Ohio.
Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis 3, Tenn.
Meeting: Statler Hotel, Boston, Mass., May 25-26 (Afternoons only), 1954.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. James M. Robb, Detroit, Mich.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Palmer House, Chicago, Ill., Oct. 11-17, 1953.

AMERICAN BOARD OF OTOLARYNGOLOGY.

Meeting: Palmer House, Chicago, Ill., Oct. 5-9, 1953.
Statler Hotel, Boston, Mass., May 25-26, 1954.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

President: Dr. Edwin N. Broyles, 1100 No. Charles St., Baltimore 1, Md.
Secretary: Dr. F. Johnson Putney, 255 So. 17th St., Philadelphia (3) Pa.
Meeting: Statler Hotel, Boston, Mass. (Afternoons) May 25-26, 1954.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Clifton E. Benson, Bremerton, Wash.
President-Elect: Dr. Carl D. F. Jensen, Seattle, Wash.
Secretary: Dr. Willard F. Goff, 1215 Fourth Ave., Seattle, Wash.

THE SECTION OF OTOLARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA.

Chairman: Dr. Victor Alfaro.
Vice-Chairman: Dr. Irvin Feldman.
Secretary: Dr. Frasier Williams.
Treasurer: Dr. John Louzan.
Meetings are held on the third Tuesday of October, November, March and May, 7:00 P.M.
Place: Army and Navy Club, Washington, D. C.

THE LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. W. L. Hughes, Lamar Life Bldg., Jackson, Miss.
Vice-President: Dr. Ralph H. Riggs, 1513 Line Ave., Shreveport, La.
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

OTOSCLEROSIS STUDY GROUP.

President: Dr. John R. Lindsay, 950 E. 59th St., Chicago 37, Ill.
Secretary: Dr. Lawrence R. Boies, Med. Arts Bldg., Minneapolis 2, Minn.
Meeting: Palmer House, Chicago, Ill., Oct. 11-17, 1953.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY.

President: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis, Ind.
President-Elect: Dr. Albert D. Ruedemann, 1633 David Whitney Bldg., Detroit 26, Mich.
Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo 2, N. Y.
Meeting: Palmer House, Chicago, Ill., Oct. 16, 1953.

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. Justo M. Alonso, Montevideo.
Executive Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa., U. S. A.
Meeting: Fourth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
President: Dr. Ricardo Tapia Acuna, Mexico City.
Time and Place: Feb. 28 to Mar. 4, 1954, Mexico City.

MISSISSIPPI VALLEY MEDICAL SOCIETY.

President: Dr. Daniel L. Sexton, St. Louis, Mo.
President-Elect: Dr. John I. Marker, Davenport, Iowa.
Secretary-Treasurer: Dr. Harold Swanberg, Quincy, Ill.
Assistant Secretary-Treasurer: Dr. Jacob E. Reisch, Springfield, Ill.
Meeting:

THE VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Peter N. Pastore, Richmond, Va.
President-Elect: Dr. G. S. Fitz-Hugh, Charlottesville, Va.
Vice-President: Dr. H. L. Mitchell, Lexington, Va.
Secretary-Treasurer: Dr. L. B. Sheppard, 301 Medical Arts Bldg., Richmond, Va.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Orwyn H. Ellis, M.D.
Secretary-Treasurer: Harold Owens, M.D.
Chairman of Section on Ophthalmology: Robert A. Norene, M.D.
Secretary of Section on Ophthalmology: Sol Rome, M.D.
Chairman of Section on Otolaryngology: Leland R. House, M.D.
Secretary of Section on Otolaryngology: Max E. Pohlman, M.D.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd., Los Angeles, Calif.
Time: 6:00 P.M., fourth Monday of each month from September to June, inclusive—Otolaryngology Section; 6:00 P.M., first Thursday of each month from September to June, inclusive—Ophthalmology Section.

THIRD LATIN AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY AND BRONCHOESOPHAGOLOGY.

President: Dr. Franz Conde Jahn.
Vice-Presidents: Drs. Julio Garcia Alvarez, Angel Bustillos and Celis Perez.
Secretary General: Dr. Victorino Marquez Reveron.
Secretary of Assemblies: Dr. Cesar Rodriquez.
Time and Place: Caracas, Venezuela, July 31, 1954.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

President: Dr. Harry Nievert, 555 Park Ave., New York (21), N. Y.
Secretary: Dr. Louis Joel Fleit, 66 Park Ave., New York (16), N. Y.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. William Banks Anderson, Durham, N. Car.
Secretary and Treasurer: Dr. Geo. B. Ferguson, Durham, N. Car.
Meeting: Charleston, S. Car., Sept. 13-16, 1953.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Clay W. Evatt, Charleston, S. Car.
Vice-President: Dr. David S. Asbill, Columbia, S. Car.
Secretary-Treasurer: Dr. Roderick Macdonald, Rock Hill, S. Car.
Meeting, Joint: Charleston, S. Car., Sept. 13-16, 1953.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President: Dr. Leland G. Hunnicutt, 98 N. Madison Ave., Pasadena, Calif.
Secretary-Treasurer: Dr. John F. Tolan, 3419 47th Ave., Seattle (5), Wash.
Meeting: Honolulu, 1954.

THE RESEARCH STUDY CLUB OF LOS ANGELES, INC.

Chairman: Dr. Isaac H. Jones, 635 S. Westlake, Los Angeles, Calif.
Treasurer: Dr. Pierre Violé, 1930 Wilshire Blvd., Los Angeles, Calif.
Program Chairmen:
Otolaryngology: Dr. Leland G. Hunnicutt, 98 N. Madison Ave., Pasadena, Calif.
Ophthalmology: Dr. Harold F. Whalman, 727 W. 7th St., Los Angeles, Calif.
Mid-Winter Clinical Convention annually the last two weeks in January at Los Angeles, Calif.

FLORIDA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Chas. C. Grace, 145 King St., St. Augustine, Fla.
President-Elect: Dr. Jos. W. Taylor, 706 Franklin St., Tampa, Fla.
Secretary-Treasurer: Dr. Carl S. McLemore, 1217 Kuhl Ave., Orlando, Fla.

THE PHILADELPHIA LARYNGOLOGICAL SOCIETY.

President: Dr. Harry P. Schenck.
Vice-President: Dr. William J. Hitschler.
Treasurer: Dr. Chevalier L. Jackson.
Secretary: Dr. John J. O'Keefe.
Historian: Dr. Herman B. Cohen.
Executive Committee: Dr. M. Valentine Miller, Dr. Charles E. Towson, Dr. Thomas F. Furlong, Dr. Benjamin H. Shuster, ex-officio.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Vice-Chairman: Dr. K. W. Cosgrove, 111 E. Capitol Ave., Little Rock, Ark.
Secretary: Dr. F. A. Holden, Medical Arts Bldg., Baltimore, Md.
Meeting:

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. James K. Stewart, Wheeling, W. Va.
Secretary-Treasurer: Dr. Frederick C. Reel, Charleston, W. Va.

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. G. C. Otrich, Belleville, Ill.
President-Elect: Dr. Phil R. McGrath, Peoria, Ill.
Secretary-Treasurer: Dr. Alfred G. Schultz, Jacksonville, Ill.

CANADIAN OTOLARYNGOLOGICAL SOCIETY SOCIETE CANADIENNE D'OTOLARYNGOLOGIE

President: Dr. D. E. S. Wishart, 170 St. George St., Toronto, Ontario.
Secretary: Dr. W. Ross Wright, 361 Regent St., Fredericton, N. B.
Place:
Time:

SOCIEDAD DE OTO-RINO-LARINGOLOGIA, COLEGIO MEDICO DE EL SALVADOR, SAN SALVADOR, C. A.

President: Dr. Victor M. Noubleau.
Secretary: Dr. Héctor R. Silva.
1o. Vocal: Dr. Salvador Mixco Pinto.
2o. Vocal: Dr. Daniel Alfredo Alfaro.

MEXICAN ASSOCIATION OF PLASTIC SURGEONS.

President: Dr. Cesar LaBoide, Mexico, D. F.
Vice-President: Dr. M. Gonzalez Ulloa, Mexico, D. F.
Secretary: Dr. Juan de Dios Peza, Mexico, D. F.

FEDERACION ARGENTINA, DE SOCIEDADES DE OTORRINOLARINGOLOGIA.

Secretario del Exterior: Dr. Juan Manuel Tato.
Sub-Secretario del Exterior: Dr. Oreste E. Bergaglio.
Secretario del Interior: Dr. Eduardo Casterán.
Sub-Secretario del Interior: Dr.Atilio Viale del Carril.
Secretario Tesorero: Dr. Vicente Carri.
Sub-Secretario Tesorero: Dr. José D. Suberviola.

ASOCIACION DE OTO-RINO-LARINGOLOGIA DE BARCELONA, SPAIN.

Presidente: Dr. J. Abello.
Vice-Presdente: Dr. Luis Suñe Medan.
Secretario: Dr. Jorge Perelló, 319 Provenza, Barcelona.
Vice-Secretario: Dr. A. Pinart.
Vocal: Dr. J. M. Ferrando.

SOCIEDAD NACIONAL DE CIRUGIA OF CUBA.

Presidente: Dr. Reinaldo de Villiers.
Vicepresidente: Dr. César Cabrera Calderin.
Secretario: Dr. José Xirau.
Tesorero: Dr. Alfredo M. Petit.
Vocal: Dr. José Gross.
Vocal: Dr. Pedro Hernández Gonzalo.

INTERNATIONAL BRONCHOESOPHAGOLOGICAL SOCIETY.

President: Dr. Andre Soulas, Paris, France.
Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa. U. S. A.
Meeting: 3rd International Congress of Broncho-Esophagology.
Time and Place: September or October, 1954, Lisbon, Portugal.

ASSOCIACAO MEDICA DO INSTITUTO PENIDO BURNIER — CAMPINAS.

President: Dr. Heitor Nascimento.
First Secretary: Dr. Roberto Barbosa.
Second Secretary: Dr. Roberto Franco do Amaral.
Librarian-Treasurer: Dr. Leoncio de Souza Queiroz.
Editors for the Archives of the Society: Dr. Guedes de Melo Filho, Dr. Penido Burnier and Dr. Gabriel Porto.

SOCIEDAD DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOSCOPIA DE CORDOBA.

Presidente: Dr. Aldo Remorino.
Vice-Presidente: Dr. Luis E. Olsen.
Secretario: Dr. Eugenio Romero Díaz.
Tesorero: Dr. Juan Manuel Pradales.
Vocales: Dr. Osvaldo Suárez, Dr. Nondier Asís R., Dr. Jorge Bergallo Yofre.

BUENOS AIRES CLUB OTORINOLARINGOLOGICO.

Presidente: Dr. Alberto P. Haedo.
Vice-Presidente: Dr. V. R. Carri.
Secretario: Dr. Renoto Segre.
Pro-Secretariò: Dr. Carlos A. Gutierrez.
Tesorero: Dr. J. M. Tato.
Pro-Tesorero: Dr. Norberto Von Soubiron.

SOCIEDAD COLOMBIANA DE OFTALMOLOGIA Y OTORRINOLARINGOLOGIA (BOGOTA, COLOMBIA).

Presidente: Dr. Alfonso Tribin P.
Secretario: Dr. Felix E. Lozano.
Tesorero: Dr. Mario Arenas A.

SOCIEDAD ESPANOLA DE OTORRINOLARINGOLOGIA.

Presidente: Dr. D. Adolfo Hinojar Pons.
Vice-Presidente: Dr. D. Jose Perez Mateos.
Secretario General: Dr. D. Francisco Marañés.
Tesorero: Dr. D. Ernesto Alonso Ferrer.

ASOCIACION DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOLOGIA DE GUATEMALA

Presidente: Dr. Julio Quevedo, 15 Calle Oriente No. 5.
First Vice-Presidente: Dr. Héctor Cruz, 3a Avenida Sur No. 72.
Second Vice-Presidente: Dr. José Luis Escamilla, 5a Calle Poniente No. 48.
Secretario-Tesorero: Dr. Horace Polanco, 13 Calle Poniente No. 9-D.

FIRST CENTRAL AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY.

President: Dr. Victor M. Noubleau, San Salvador.
Secretary-Treasurer: Dr. Hector R. Silva, Calle Arce No. 84, San Salvador, El Salvador, Central America.

THIRD LATIN-AMERICAN CONGRESS OF OTORINOLARINGOLOGIA.

President: Dr. Franze Conde Jahn, Caracas.
Secretary: Dr. Victorino Marquez Reveron, Caracas.
Meeting: Caracas, Venezuela, Feb. 21-25, 1954.

CONTENTS